

SHOULDER DYSTOCIA AT DELIVERY: POPULATION-BASED STUDIES OF RISK FACTORS

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LIST OF PAPERS

Paper I

Overland EA, Spydsaug A, Nielsen CS, Eskild A.

Risk of shoulder dystocia in second delivery: does a history of shoulder dystocia matter?

American Journal of Obstetrics and Gynecology 2009; 200:506.e1-6.

Paper II

Overland EA, Vatten LJ, Eskild A.

Risk of shoulder dystocia: associations with parity and offspring birthweight. A population study of 1 914 544 deliveries.

Acta Obstetrica et Gynecologica Scandinavica 2012; 91:483-488.

Paper III

Overland EA, Vatten LJ, Eskild A.

Pregnancy week at delivery and the risk of shoulder dystocia: a population study of 2 014 956 deliveries.

BJOG: An International Journal of Obstetrics & Gynecology 2014; 121(1):34-42.

ABBREVIATIONS

BMI	Body mass index
CI	Confidence interval
MBRN	Medical Birth Registry of Norway
OR	Odds ratio
aOR	Adjusted odds ratio
cOR	Crude odds ratio
RR	Relative risk
SPSS	Statistical Package for Social Science

1.0 INTRODUCTION

Shoulder dystocia is described as the failure of delivery of the fetal shoulders after delivery of the fetal head (1-6). Shoulder dystocia can be a dramatic event; the phrase “the nightmare of the obstetrician” is often used to describe the severity of this emergency obstetric situation (4, 7). The need for resolute action to avoid a potential fatal outcome for the offspring underlines the drama. Knowledge of the risk factors for shoulder dystocia is necessary in order to prevent and accurately manage the condition.

Shoulder Dystocia

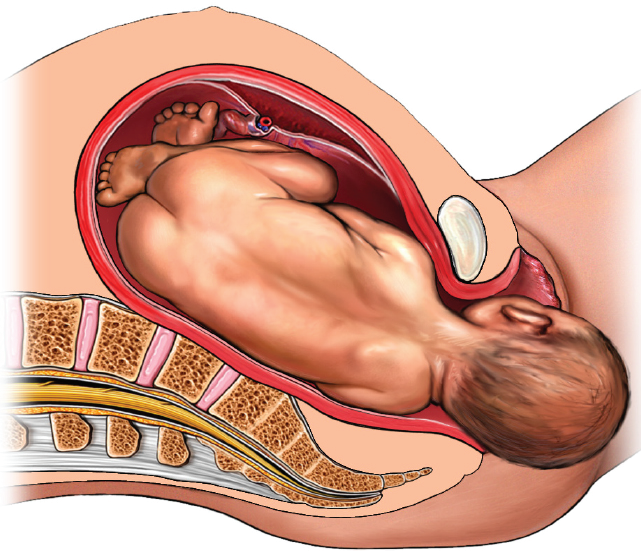


Figure 1. Shoulder dystocia. Used with permission © Nucleus Media 2013

1.1 What is shoulder dystocia?

The mechanism of normal delivery

To understand the mechanisms of shoulder dystocia, it is necessary to understand the mechanisms of a normal delivery. A normal vaginal delivery depends on the interplay between the pelvis, the uterine contractions and the fetus (8). Because of the anatomy of the human maternal pelvis, the fetal head needs to change positions while descending through the birth canal (Figure 2).

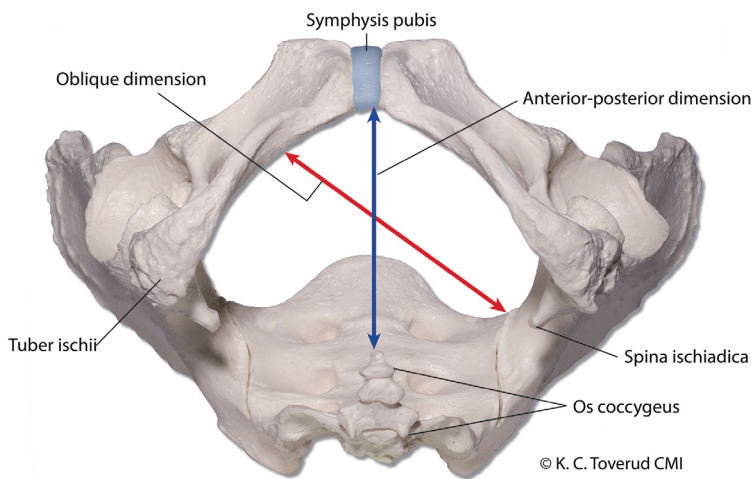
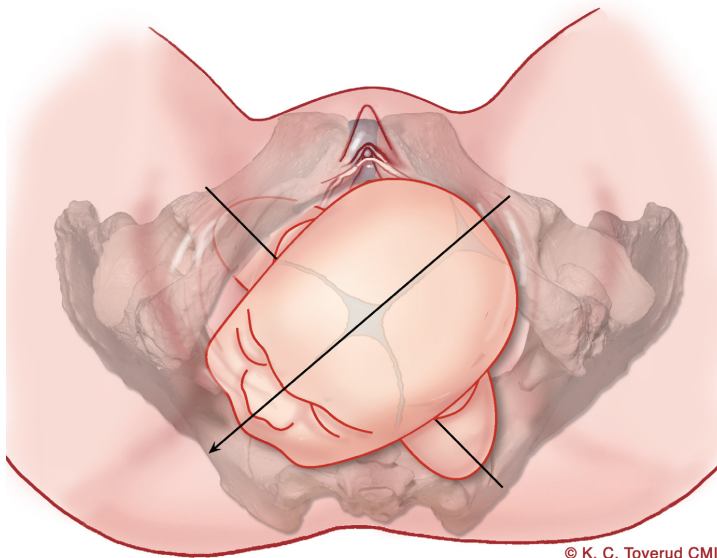


Figure 2. The human maternal pelvis viewed from below. Used with permission © K. C. Toverud CMI 2013

These adjustments of the fetal head through the birth canal, are called the cardinal movements (1): Flexion: The fetal head flexes so that the fetal chin is brought down towards the fetal thorax and ensures that the shortest diameter of the fetal head passes through the pelvic inlet in the most favorably way. Internal rotation: The fetal head gradually turns so that the occiput moves towards the maternal symphysis pubis anteriorly or less commonly, posteriorly towards the sacrum. Extension: The head reaches the pelvic outlet and undergoes extension during the expulsion of the head through the vulvar opening. The fetal head has now turned 90 degrees towards the occiput anterior (or posterior) position by the time it reaches the outlet. As the planes of the pelvic inlet and outlet are skew rather than parallel, the path between the inlet and the outlet has a curved architecture rather than a straight one. External

rotation: The delivered head next undergoes restitution, and the occiput turns toward either the left or the right maternal tuberosity.

The fetal shoulder normally passes through the oblique dimension of the pelvic inlet and the anterior shoulder can thereafter slide underneath the maternal symphysis (Figure 3). The shoulders follow while the head has completed its external rotation. Normally, at this point the shoulders have already moved through the pelvic inlet and passes through the birth canal during the delivery process. The shoulders thereafter pass the pelvic outlet and the vulvar opening in an adducted, oblique position. All of these movements normally take place during uterine contractions.



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Figure 3. Normal delivery. The shoulders pass through the oblique dimension of the pelvic inlet. Used with permission © K. C Toverud CMI 2013

Pathophysiology of shoulder dystocia

Shoulder dystocia can be characterized as a delayed or difficult delivery of the fetal shoulders following the delivery of the fetal head (9-11). Shoulder dystocia may occur when the fetal shoulders do not pass through the oblique dimension of the pelvic inlet, which is the most favorable, but instead pass through the anterior-posterior dimension, which is smaller (2) (Figure 2). If the shoulders enter the pelvic inlet through the anterior-posterior dimension this may cause impaction of the anterior shoulder above the symphysis. Impaction of the posterior shoulder on the sacral promontory can also happen, but is probably rarer (1). The result of both anterior and posterior impaction is a mechanical obstruction caused by a discrepancy in the size of the pelvic inlet and the fetal shoulders. This impaction of the anterior, the posterior or both shoulders above the pelvic inlet causes shoulder dystocia (2, 4).

There may be a number of explanations why shoulder dystocia occurs. Some authors have postulated that natural rotation is prevented when friction between the fetus and the vaginal walls is increased, therefore forcing the shoulders to pass through the anterior-posterior dimension of the pelvic inlet, resulting in shoulder dystocia (2). Theoretically, the lack of sufficient rotation of the shoulders may also happen when the delivery process is enforced or when the delivery process goes particularly fast. Soft tissue obstruction of the fetal shoulders may be another mechanism that can lead to difficult delivery of the fetal shoulders.

Diagnostic criteria for shoulder dystocia

Shoulder dystocia is described as a delayed or difficult delivery of the fetal shoulders after the delivery of the fetal head, still the diagnosis of shoulder dystocia remains based on the subjective recognition of the condition by the birth attendant.

There is a lack of uniform diagnostic criteria for shoulder dystocia. Some authors regard any general difficulties in extracting the shoulders after the delivery of the head as shoulder dystocia (10, 11). Others argue the diagnosis should only be made when the normal gentle downward traction of the fetal head does not result in the delivery of the shoulders, and the clinical situation requires additional obstetric maneuvers (7, 12-16). Retraction of the fetal head towards the vagina may be present in shoulder dystocia, and is considered a clinical sign of the condition, commonly called the “turtle sign”.

Some authors have advocated for more objective, time-based criteria for the diagnosis of shoulder dystocia. A study by Spong et al, published in 1995 included 250 unselected deliveries, and the head-to-body delivery intervals were timed (17). The mean head-to-body delivery interval for normal deliveries was 24 seconds compared to 79 seconds in deliveries that was diagnosed with shoulder dystocia. The authors proposed that a head-to-body delivery interval of more than 60 seconds should define the diagnoses of shoulder dystocia. Beal et al later validated this timing criteria (18). However, this criterion for shoulder dystocia based on head-to-body delivery interval has been criticized (19). To illustrate: a pause between the uterine contractions after the delivery of the fetal head may result in a delayed delivery of the shoulders. Is such a situation a shoulder dystocia? If the birth attendant, in this situation, chooses to enforce the delivery of the shoulders to reduce the head-to-body delivery interval, by performing a hard traction of the fetal head downwards or by using additional maneuvers to deliver the shoulders, the delivery may be experienced as difficult and thus fulfill one of the suggested criteria of shoulder dystocia. However, in doing this, the birth attendant might cause an “iatrogenic” difficult delivery of the shoulders, and the forceful traction of the head may cause offspring morbidity (20). On the other hand, in such a situation, many obstetricians and midwives will argue that instead of urging the delivery of the shoulders, it is best to wait for a new contraction to deliver the shoulders and body (19). This latter approach is commonly called “the two step approach” (21). If “the two step approach” is followed, the interval between the delivery of the head and the delivery of the whole body (head-to-body delivery interval) may be longer than 60 seconds. This situation may be regarded as a shoulder dystocia by the “time-based” criterion, but it is questionable if this situation should be reported as a “true” shoulder dystocia.

1.2 History

Shoulder dystocia is a dramatic obstetric situation which has been described in the medical literature for more than three centuries. An early written description of shoulder dystocia is from 1652, in a thesis by Bourgeois (22). Other historic descriptions were made by De la Mott in and by Smellie (23, 24). The latter is a case report of a dramatic obstetric situation. Smellie writes: “...a sudden call to a gentlewoman in labor. The child’s head delivered a long time- but even with hard pulling from the midwife, the remarkably large shoulder prevented delivery. I have been called by midwives to many cases of this kind, in which the child was frequently lost” (24) .

Jean Marie Jacquemer (1806-1879) was a pioneer in the history of shoulder dystocia (23). In 1848 he had to deal with a case of the condition. After one hour he managed to deliver the child, who weighed 5500 grams. Apparently the child recovered and survived. He described a series of maneuvers that he performed in an attempt to deliver the child, but eventually he performed an extraction of the posterior arm, which resolved the situation. In 1860, Jacquemer published a paper in the *Gazette Hébdomadaire de Médecine et Chirurgie*, in which he described the different techniques to resolve shoulder dystocia. This work was based on 26 observations described in literature at that time, and in Europe the maneuver of extracting the posterior arm was called the Jacquemer's maneuver after this giant in French obstetrics (23). However, in English scientific literature this maneuver was described by Barnum in a paper published in 1945, and is now commonly known as Barnum's maneuver (25).

In 1899, Bonneaire and Buè introduced a maneuver with hyperflexion of the maternal hips against the maternal abdomen to resolve shoulder dystocia (23). However, it was not until the early 1980s that this maneuver was rediscovered and named by Gonik et al as the McRobert's maneuver (26). Apparently this maneuver was demonstrated and used at the University of Texas Medical School by Dr William A. McRoberts, Jr.

Another classical work was done by Woods and Westbury. In 1942, at the Obstetrics and Gynecology meeting of the New York Academy of Medicine, they presented a principle of physics in resolving shoulder dystocia (27). They demonstrated how to release the impacted anterior shoulder by progressively rotating the posterior shoulder 180 degrees in a corkscrew fashion. This work was published in 1943 and the maneuver is commonly known as the Wood's corkscrew maneuver.

The highly regarded Norwegian obstetrician Jørgen Løvset (1896-1981) was engaged in many fields of the obstetric profession, and management of shoulder dystocia was among them. In his classic text books "Lærebok i obstetikk for jordmødre", first published in 1959 and "Vaginal operative delivery", first published in 1968, he describes a maneuver to resolve shoulder dystocia by hooking one or two fingers into the fetal axillary fold and perform a rotation (28, 29). This procedure is still in use in Norway, and is at many maternity wards regarded as the first choice for resolving shoulder dystocia.

1.3 Occurrence

Table 1 presents the reported occurrence of shoulder dystocia in different studies. As shown in the table, the reported occurrence of shoulder dystocia varies considerably across studies: from 0.13% to 13.7% of all vaginal deliveries (Table 1).

This variation may have different explanations:

- 1) The definitions of shoulder dystocia used in the different studies may vary.
- 2) The reporting routines may vary.
- 3) The selection of the study samples may vary.
- 4) The estimated occurrence of shoulder dystocia may be uncertain due to lack of statistical power.
- 5) There may be differences in the use of cesarean delivery among women with high risk of shoulder dystocia and shoulder dystocia occurs in vaginal deliveries only.
- 6) There may be a true difference in the occurrence of shoulder dystocia.

Table 1. *Reported occurrence of shoulder dystocia in different studies*

Author	Country	Publication year	Number of deliveries	Cases with shoulder dystocia (%)
Swartz DP (30)	USA	1960	20 599	0.15
Benedetti TJ et al (9)	USA	1978	8890	0.37
Hopwood H (11)	USA	1982	17 735	0.5
Acker D et al (31)	USA	1985	14 721	2.1
Gross SJ et al (32)	Canada	1987	10 662	0.2
Langer O et al* (7)	USA	1991	75 979	0.6
Nocon JJ et al (33)	USA	1993	12 532	1.4
Baskett TF et al (34)	Canada	1995	40 518	0.6
Spong CY et al (17)	USA	1995	250	16
Geary M et al (35)	Ireland	1995	10 468	0.6
Gherman R et al (14)	USA	1997	44 072	0.6
Nesbitt TS et al (36)	USA	1998	175 886*	3.0
Beall MH et al (18)	USA	1998	722	13.7
Kees S et al (37)	Israel	2001	24 000	0.23
Stallings SP et al (38)	USA	2001	8282	1.67
Al HM et al (39)	Ireland	2001	9541	0.44
Christofferson M et al (40)	Sweden	2002	1 076 545	0.13
Ouzounaian JG et al (41)	USA	2005	267 228	0.6
MacKenzie IZ et al (42)	UK	2007	79 781	0.6

Christie L et al (43)	Jamaica	2008	8267	0.83
Moore H et al (44)	USA	2008	1 126 593	2.3
Fadl HE et al (45)	Sweden	2010	1 260 297	0.2 (without gestational diabetes) 0.6 (with gestational diabetes)
Dodd JM et al (46)	Australia	2012	1 14 827	1.1

* Offspring of more than 3500 grams only

1.4 Complications

Shoulder dystocia is associated with an increased risk of maternal morbidity and an increased risk of offspring morbidity and mortality.

Maternal complications

Soft tissue injuries, including third- and fourth-degree tearing of the sphincter ani muscle and vaginal lacerations, are the most common maternal complications in deliveries with shoulder dystocia (4, 47). Excess postpartum hemorrhage due to soft tissue tearing or to uterine atony may also occur. It is reported that among deliveries with shoulder dystocia, 11% of women experienced excess postpartum hemorrhage (>500 ml), and up to 4% experienced a fourth-degree lacerations of the sphincter ani muscle (14, 48, 49). Serious maternal complications, such as symphysis diathesis and uterine rupture, have also been reported (50). It is unclear if these maternal complications are due to the management of shoulder dystocia, or to the condition itself.

Offspring complications

Serious complications for the offspring can occur as a consequence of shoulder dystocia or its management, including death (2, 19, 49, 51, 52).

Brachial plexus palsy

The brachial plexus is the major nerve network supplying the upper limb. Palsy in the limbs caused by injury of the brachial plexus is one of the most common serious offspring complications of shoulder dystocia and is reported to occur in 3.3% to 32% of all cases (2, 15, 31, 32, 39, 53-57). The mechanism behind brachial plexus palsy is overstretching of the nerves in the brachial plexus. The overstretching may be caused by the delivery itself, or it may be due to hard exogenous traction of the fetal head downwards during the management of shoulder dystocia (34, 51, 58-60). The injury may be localized to the upper part of the plexus (erb's palsy), or to the lower part. Erb's palsy is a result of injury to the spinal nerves C5-C6, and sometimes C7. This injury causes a hanging of the upper limb that may extend to the elbow. If also the spinal nerves C7-T1 are involved, the injury will also include palsy of the hand, causing a clawhand deformity (1). The majority of the offspring diagnosed with brachial plexus palsy will recover within 3 months of birth, but about 10% will suffer from permanent palsy (14, 15, 31, 61, 62). It has been reported that up to half of the infants with brachial plexus palsy had births without the occurrence of

shoulder dystocia (12, 14, 15) Interestingly, the occurrence of brachial plexus palsy varies between countries: 1.3/1000 live born infants in the USA and Sweden, but only 0.4/1000 live born infants in the United Kingdom and Ireland are reported to have brachial plexus palsy (57, 63, 64).

Fractures

Fractures of the offspring's humerus or clavicle may also occur (39). Gherman et al (1998) found that 9.5% of shoulder dystocia cases were complicated with fractures of the clavicle, and 4.2% were complicated with fractures of the humerus (15). Again, it is not clear whether these complications occur as a result of the management of shoulder dystocia or of the condition itself.

Cerebral hypoxia

Shoulder dystocia may cause asphyxia leading to hypoxic ischemic encephalopathy, which may cause permanent brain damage and even offspring death (15, 38, 52). The determining factor of the development of birth asphyxia is the head-to-body delivery interval (65, 66). However, the underlying pathophysiological mechanisms of hypoxic ischemic encephalopathy in deliveries with shoulder dystocia are still unclear. Two possible mechanisms have been suggested:

- a) Compression of the umbilical cord between the vaginal wall and the offspring.
- b) Compression of the offspring's carotid vessels caused by the compression of the offspring's neck against the maternal perineum.

Shortly after a delivery complicated with shoulder dystocia, it is recommended to perform a clinical judgment of the offspring including an Apgar scoring and also a measurement of the umbilical cord arterial pH and base excess (67). The latter is regarded as a measurement of the offspring's central acidosis. Wood et al performed an often-cited study published in 1973, which reported that the decline in umbilical artery pH was as fast as 0.14 per minute of head-to-body delivery interval (68). Such rapid decline in umbilical artery pH indicates that fetal asphyxia can develop quickly when there is a delayed delivery of the shoulders. This study has been used to argue for an enforced delivery of the shoulders after the delivery of the head. However, this study has been criticized due to the limited study sample (22 cases only) and the fact that none of the deliveries were affected by shoulder dystocia (38).

Leung et al performed a study published in 2010 including 200 shoulder dystocia cases and found that the umbilical arterial pH declined at a rate of only 0.011 per minute of head-to-body delivery interval (65). They concluded that the risk of acidosis, determined by measuring umbilical arterial pH, is very low when the head-to-body delivery interval does not exceed 5 minutes.

If asphyxia due to shoulder dystocia is caused by the compression of the umbilical cord between the vaginal wall and the offspring, a positive correlation between the degree of umbilical artery acidosis and the risk of developing hypoxic ischemic encephalopathy would be expected. However, there are reported cases of severe morbidity caused by shoulder dystocia even when umbilical arterial pH does not indicate acidosis (65). This might suggest that the most likely explanation of hypoxic ischemic encephalopathy due to shoulder dystocia is the compression of the offspring's carotid vessels. This compression may affect not only the supply of oxygenated blood to the brain (the arteries), but also reduce the ability to eliminate the products of anaerobic metabolism (the veins). Consequently, umbilical arterial pH may not always reflect the degree of cerebral hypoxia (65). When shoulder dystocia occurs during delivery, a clinical judgment including an Apgar score might be a more adequate indicator of cerebral hypoxia.

Regardless of the different theories of pathophysiology, the critical time interval before shoulder dystocia causes offspring morbidity or mortality due to cerebral hypoxia has yet to be determined. However, studies have suggested that if the head-to-body delivery interval is less than 5 minutes the risk of hypoxic ischemic encephalopathy is very low (69).

1.5 Management

Management of mothers at risk for shoulder dystocia

Shoulder dystocia is regarded as an unpreventable situation (70). However, conditions associated with some of the risk factors for shoulder dystocia may be preventable.

High maternal body mass index (BMI), extensive weight gain during pregnancy and maternal diabetes, including gestational diabetes, are factors associated with a high offspring birthweight, and high offspring birthweight is associated with increased risk of

shoulder dystocia (chapter 1.6). A large meta-analysis on the effect of intervention (dietary interventions, lifestyle interventions based on physical activity, or both) on adverse obstetric outcomes, among others shoulder dystocia, was published in 2012 (71). Four of the total of 44 trials studied the effect on shoulder dystocia (n= 2317 women) (72-75). In a meta-analyses of these four studies, the overall risk of shoulder dystocia was reduced by 61% in the intervention group as compared to the control group (relative risk (RR) 0.39, 95% confidence interval (CI) 0.22-0.70; p=0.002). Dietary interventions were associated with greater reduction in maternal weight gain and occurrence of diabetes as compared to lifestyle intervention based on physical activity or a mixed approach. Also, the results suggested that overweight and obese women benefitted from these interventions more than normal-weight women. The beneficiary effect of the intervention on the risk of shoulder dystocia may be a result of a reduced occurrence of high offspring birthweight and reduced occurrence of gestational diabetes in the intervention group.

A meta-analysis of effects of treatment in women with gestational diabetes was published in 2010 (76). Five randomized controlled trials were included, in which intervention consisted of lowering blood glucose, either alone, or in combination with specialized antenatal care. Two out of five trials reported on shoulder dystocia (72, 73). In women with gestational diabetes receiving specialized antenatal care during pregnancy, shoulder dystocia was significantly less common (odds ratio, OR 0.40, 95% CI 0.21-0.75) compared to women with gestational diabetes not receiving specialized antenatal care.

A Cochrane review published in 2001, assessed the effect of a policy of elective delivery, as compared to expectant management, in term pregnancies of diabetic women on the occurrence of shoulder dystocia, among other maternal and perinatal adverse outcomes (77). One trial only, was included in the review (78). This trial included 200 insulin-requiring diabetic women, and compared induction of labor at 38 completed weeks of pregnancy to expectant management up to 42 completed weeks of pregnancy. The risk of macrosomia, defined as offspring birthweight of >4000 grams, was significantly reduced in the induction group (RR 0.56, 95% CI 0.32-0.98). Three deliveries with shoulder dystocia were reported in the expectant management group, as compared to no cases in the induction group. The difference was non significant (RR 0.14, 95%CI 0.01-1.73). The authors concluded that the limited number of women included in the study did not permit firm conclusions to be drawn.

Guidelines/recommendations to reduce the risk of shoulder dystocia in different countries

- Guidelines from UK (2012)

Induction of labor of diabetic women at term with suspected development of large offspring is recommended. Induction of labor of non-diabetic women with estimated large offspring is not recommended (79-82). Elective cesarean delivery of diabetic mothers should be considered when the estimated offspring weight is 4500 grams or more (83).

<http://www.rcog.org.uk/womens-health/clinical-guidance/shoulder-dystocia-green-top-42>

- Guidelines from the USA (2002)

Elective induction of labor or elective cesarean delivery for all women suspected of carrying a fetus with macrosomia is not appropriate. Planned cesarean delivery to prevent shoulder dystocia may be considered for suspected fetal macrosomia with estimated fetal weights exceeding 5000 grams in women without diabetes and 4500 grams in women with diabetes (6).

http://journals.lww.com/greenjournal/Abstract/2002/11000/ACOG_Practice_Bulletin_No_40_Shoulder_Dystocia.42.aspx

- Guidelines from Denmark (2007)

Elective cesarean delivery of diabetic mothers with an offspring with an estimated birthweight of more than 4500 grams may be considered.

<http://dsog.dk/wp/guidelines-2/obstetrik>

- Guidelines from Norway (2013)

Elective cesarean delivery of diabetic mothers, carrying an offspring with estimated birthweight of more than 4500 grams may be considered. Elective cesarean delivery of mothers with a previous occurrence of shoulder dystocia, carrying an offspring with estimated birthweight of more than 4500 grams, may be considered. Close follow-up and treatment of diabetic women is recommended. Induction of labor for non-diabetic women carrying large offspring is not recommended.

<http://legeforeningen.no/Fagmed/Norsk-gynekologisk-forening/Veiledere>.

Ahead of print, February 2014.

Management of shoulder dystocia

Since shoulder dystocia is difficult to predict, clinicians must be prepared for this condition to occur during any vaginal delivery (33). It is recommended to be well prepared so that resolute and step-wise actions can be taken to resolve the problem as safely and effectively as possible without causing unnecessary harm or trauma (13, 84-87). A systematic approach and collaboration between colleagues are recommended in order to solve the emergency situation caused by shoulder dystocia properly. It is recommended that birth attendants at obstetrical departments undergo regular training in clinical management of shoulder dystocia (67, 88-91). Several studies have demonstrated improvement in the management of shoulder dystocia after training (88, 91, 92). The occurrence of adverse neonatal outcomes such as brachial plexus palsy also decreased significantly after such training (93, 94).

Internationally there is considerable agreement regarding the management of shoulder dystocia (6, 67):

Management of shoulder dystocia

1. Call for additional help and assistance.
2. Perform the McRoberts maneuver: A sharp flexion of the maternal thighs against the maternal abdomen. This will increase the functional diameter of the maternal pelvis and might resolve the situation (14, 16, 26, 59).
3. Apply a suprapubic pressure on the fetal anterior shoulder from the dorsal side of the fetus. Such pressure may result in an adduction of the fetal shoulders, and at the same time the pressure may push the shoulders into a position which corresponds to the oblique dimension of the pelvis inlet.
4. Perform a rotation of the shoulder to correspond to the oblique dimension of the pelvic inlet. To do this, the birth attendant has to gain access to the vagina to attempt to rotate the shoulders. Various manual maneuvers to rotate the shoulders via the vagina are described in the literature: the Rubin's maneuver, the Wood's screw maneuver or the Løvset's delivery maneuver of the shoulders after the delivery of the head. (27, 28, 95). However, no single maneuver has proven superior to the others (96, 97).
5. Perform an extraction of the fetal posterior arm (the Barnum's maneuver) (98).
6. Avoid fundal pressure and active maternal pushing (32, 51).
7. Avoid excessive downward traction of the offspring's neck (20, 34, 59, 86, 99).

Episiotomy does not seem to resolve the problem of shoulder dystocia, but may be useful since it generates more space to undertake the above-mentioned obstetric maneuvers via the vagina (100, 101).

Various other procedures to manage shoulder dystocia include:

- Placing the mother on her hands and knees (All-fours technique or the Gaskin's maneuver) (48, 102, 103).
- Splitting the symphysis of the maternal pelvis to relieve the obstructed fetal shoulder (50, 104). This maneuver is associated with high risk of maternal morbidity (105).
- Replacement of the offspring's head into the pelvis followed by a cesarean delivery (the Zavanelli maneuver) (106). Even though the success of this maneuver is reported to be high, it is likely that the reporting is biased, as a successful outcome is more likely to be reported than an unsuccessful outcome. The risk of maternal morbidity for this maneuver is also probably high (107, 108). In addition, at the stage at which this maneuver is considered, a large proportion of offspring is likely to have either irreversible hypoxia, or is already dead.

These various other procedures listed above, are mainly described as case reports. For practical and ethical reasons it is close to impossible to study and compare the effects of the various procedures by randomized controlled studies.

1.6 Known and potential risk factors for shoulder dystocia

Some maternal, offspring and intrapartum factors have been identified as known or potential risk factors for shoulder dystocia. A risk factor is defined as a factor associated with an increased risk of the outcome (109).

Maternal risk factors

Diabetes mellitus

Maternal diabetes, including diabetes type 1, type 2 and gestational diabetes increase the risk of shoulder dystocia. The risk is reported to increase by two- to six-fold as compared to non-diabetic pregnancies, independent of offspring birthweight (36, 45, 62, 110, 111). Offspring born to diabetic mothers tend to have a larger body circumference in relation to the head circumference compared to offspring born to non-diabetic mothers. Such body configuration may increase the risk of shoulder dystocia (7, 112-115).

Obesity and excessive weight gain during pregnancy

Maternal obesity and excessive weight gain during pregnancy increase the risk of having a high birthweight offspring. Since high offspring birthweight is strongly linked to shoulder dystocia, maternal obesity and excessive weight gain during pregnancy are therefore likely to increase the absolute risk of shoulder dystocia (116, 117). However, studies on maternal obesity as an independent risk factor show conflicting results. In 1998 Sama et al performed a case-control study that included 62 shoulder dystocia cases. They concluded that shoulder dystocia is associated with high maternal bodyweight. A year later, however, Gerner et al published a case-control study that did not find any association between maternal BMI and shoulder dystocia (118, 119). In 2003 Robinson et al published a case-control study and the authors concluded that maternal obesity (>91 kg) was not an independent risk factor for shoulder dystocia after adjusting for confounding variables (120). Leung et al also suggested that, after adjustment for confounding factors such as birthweight, high maternal BMI was not associated with an increased risk of shoulder dystocia (121). Jensen et al published a large retrospective cohort study in 2003. They found a higher occurrence of high offspring birthweight in obese women, but obesity was not found to be an independent risk factor for shoulder dystocia (122). In 2004, Cedergren performed a prospective population-based cohort study on the risk of shoulder dystocia in 3480 morbidly obese women ($\text{BMI} >40 \text{ kg/m}^2$) and 12 698 obese women (BMI of 35-40 kg/m^2) as compared to normal weight women (BMI 19.8-26 kg/m^2) (123). The results suggested that maternal obesity, both morbid obesity and obesity, is associated with pregnancy complications such as shoulder dystocia. However, adjustment for offspring birthweight was not made. In 2012 Gilead et al published a population-based cohort study to compare the pregnancies of obese, defined as pre pregnancy BMI of 30 kg/m^2 or higher, and non obese patients. They found no significant difference in the risk of shoulder dystocia among the obese and non obese patients (124).

Thus, there are conflicting results in studies on the association of maternal obesity with shoulder dystocia, and some studies lack statistical power to detect true associations. In general maternal obesity seems to be associated with an increased risk of shoulder dystocia in crude analyses, but after adjustment for offspring birthweight, the results suggest that maternal obesity is not an independent risk factor for shoulder dystocia.

Advanced maternal age

The occurrence of shoulder dystocia seems to increase with increasing maternal age. However, factors associated with high offspring birthweight like multiparity and high maternal BMI are more common among mothers giving birth at an advanced age (125-129). Thus, the increased risk of shoulder dystocia in mothers at advanced age may have been confounded by multiparity and high maternal BMI. Also, the increased prevalence of diabetes in mothers at advanced age, may confound the positive association of maternal age and the occurrence of shoulder dystocia (7)

Offspring risk factors

High offspring birthweight

Numerous studies have shown that high offspring birthweight is strongly associated with the risk of shoulder dystocia (Table 2). In fact, high offspring birthweight is regarded as a major risk factor for shoulder dystocia. However, only a few prior studies have had sufficient statistical power to investigate the association of very high birthweights with shoulder dystocia. An offspring weight of 4500 grams or more is commonly regarded as a macrosome offspring. However, although macrosome offspring are strongly associated with an increased risk of shoulder dystocia, the majority of deliveries of macrosome offspring are not affected by shoulder dystocia.

Table 2. *Offspring birthweight and the risk of shoulder dystocia*

Author	Country	Publication year	Number of deliveries	Number of shoulder dystocia	Association
Swartz DP (30)	USA	1960	20 599	31	Increasing occurrence of shoulder dystocia by increasing birthweight. The overall occurrence was 0.15%. When birthweight was > 4000 grams the occurrence was 1.7%
Benedetti T et al (9)	USA	1978	8890	33	Increasing occurrence by increasing birthweight. The occurrence was 1.6% when birthweight was < 4000 grams. When birthweight was > 4000 grams the occurrence was 23%.
Acker DB et al (31)	USA	1985	14 721	309	Increasing occurrence by increasing birthweight among women with and without diabetes. For the total study population the occurrence was 0.2% when the birthweight was 2500-3000 grams. Increasing to 23.9% when birthweight was > 4500 grams. Recommends cesarean delivery for diabetic women carrying a fetus with estimated birthweight of more than 4000 grams.
Sandmire HF et al (130)	USA	1988	73 cases, 146 controls		Case control study. Fetal macrosomia (> 4000 grams and > 4500 grams) was associated with shoulder dystocia.
Gross SJ et al (32)	Canada	1987	10 662	91	60% of all cases of shoulder dystocia occurred when the fetal birthweight was > 4 kg.

Gross TL et al (131)	USA	1987	7013	49	Increasing occurrence of shoulder dystocia by increasing birthweight. The occurrence was 0.5% when the birthweight was <2500 grams, increasing to 35% when the birthweight was \geq 4500 grams.
Rydhstrom H et al (132)	Sweden	1989	110 (\geq 5700 grams)	44	40% occurrence of shoulder dystocia when the birthweight was \geq 5700 grams.
Langer O et al* (7)	USA	1991	75 979	456	Strong association between birthweight and risk of shoulder dystocia. OR for shoulder dystocia was 305 when the birthweight was \geq 5000 grams when 3500-3750 grams was the reference.
Baskett TF et al (99)	Canada	1995	40 518	254	The occurrence of shoulder dystocia was 0.35% when the fetal weight was <4000 grams, increasing to 20% when the fetal weight was >5000 grams.
Lewis DF et al (133)	USA	1998	99 cases, 1523 controls		Case control study. Cases had higher birthweight than the controls and suggests that macrosomia (>4000 grams) was the only variable factor associated with increased risk of shoulder dystocia.
Ouzounaian JG et al* (41)	USA	2005	267 288	1 686	OR for shoulder dystocia was 8.5 when birthweight was >4000 grams, increasing to 11.3 when birthweight was >4500 grams compared to birthweight <4000 grams among women without diabetes.

Sheiner E et al * (134)	Israel	2006	107 965	245	Strong association of birthweight with risk of shoulder dystocia. Adjusted OR for shoulder dystocia 24.3 when birthweight was >4000 grams (birthweight < 4000 grams as the reference).
Mansor A et al* (135)	Malaysia	2010	899 cases (>3.5kg)	36	Macrosomia (> 4000 grams) was associated with increased risk of shoulder dystocia after adjustment for other study factors.
Gupta M et al (136)	UK	2010	40 284	240	The occurrence of shoulder dystocia increased by increasing birthweight: 0.1% when the birthweight was <3000 grams increasing to 10.3% when the birthweight was >4500 grams.
Bjørnstad AR et al* (137)	Norway	2010	304 968	1.2%	The risk of shoulder dystocia was strongly associated with increasing birthweight. OR was 0.43 when the birthweight was 0.43 increasing to 64.2 when the birthweight was >5000 grams.
Vidarsdottir H et al* (138)	Iceland	2011	343 cases, 686 controls		The risk of shoulder dystocia was strongly associated with macrosomia (>5000 grams). OR for shoulder dystocia was 26 for offspring weighing >5000 grams when comparing with normal weighing offspring.
Tsur A et al* (139)	Israel	2012	240 189	451	Increasing birthweight was strongly associated with increasing risk for shoulder dystocia. OR was 16 for shoulder dystocia when birthweight >4000 grams compared to birthweight < 4000 grams.

Dodd M et al * (46)	Australia	2012	114 827	1303	Macrosomia (>4000 grams) was a significant risk factor for shoulder dystocia: aOR for shoulder dystocia to be 6.16 when birthweight was >4500 grams and aOR 12.7 when birthweight was >4500 grams.
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* Data analyzed by multivariable models.

Intrapartum risk factors

Labor abnormalities

Potential risk factors for shoulder dystocia include labor abnormalities such as prolonged or precipitous labor. Labor abnormalities have been studied, but the estimated risks associated with shoulder dystocia are conflicting. Some studies have shown a higher occurrence of prolonged and precipitous labor among deliveries with shoulder dystocia compared to deliveries without shoulder dystocia, but others have found no association between prolonged labor and increased risk of shoulder dystocia (9, 16, 99, 118, 131, 140-143).

Operative vaginal delivery

Operative vaginal delivery seems to be associated with shoulder dystocia. However, it is unclear if the operative delivery is an independent risk factor, or if it is the underlying condition that causes the operative delivery, that is associated with shoulder dystocia (9, 16, 99, 142, 144, 145).

Vacuum-assisted deliveries seem to have a higher risk of shoulder dystocia than forceps-assisted deliveries. It is unclear if vacuum assistance is more often used in abnormal deliveries with a high risk of shoulder dystocia compared to forceps-assisted deliveries, or if vacuum assistance is stronger associated with shoulder dystocia than forceps assistance. However, in a randomized controlled study Bofill et al demonstrated a higher rate of shoulder dystocia in vacuum-assisted deliveries than in forceps-assisted deliveries (4.7% versus 1.9%) (146).

A study by Benedetti and Gabbe published in 1978, concluded that the combination of a macrosome offspring, prolonged second stage of labor and a midpelvic operative delivery is associated with an increased risk of shoulder dystocia (9). This has also been suggested in other studies (36).

Epidural analgesia

A case-control study performed by Christoffersson et al, published in 2003, used data from patient records at four large hospitals in Sweden. The authors concluded that epidural analgesia is an independent risk factor for shoulder dystocia (OR 1.89, 95% CI 1.07-3.34) (57).

1.7 Background for the present studies

It is often claimed that most shoulder dystocia cases occur without any known underlying risk factors. Thus, our knowledge of the risk factors for shoulder dystocia is still insufficient (2).

Shoulder dystocia is one of the most dramatic obstetric emergency situations that can happen at delivery. Because of the potential fatal outcome for the offspring and the physical and emotional trauma this situation may cause for the mother, reliable knowledge about risk factors is necessary. Shoulder dystocia is a rare event. To ensure sufficient statistical power, large study populations are necessary to gain reliable estimates of the risk of shoulder dystocia.

Background for Paper I

History of shoulder dystocia and risk of recurrence

A history of shoulder dystocia is assumed to be a risk factor for shoulder dystocia in a subsequent pregnancy. Therefore many clinicians practice the principle of “once a shoulder dystocia always a cesarean” (147). The impact of shoulder dystocia during the first delivery on the risk of shoulder dystocia in the second has, to our knowledge, been reported in five previous studies, in which the reported estimates of recurrent risk varied widely, from 1% to 14% (99, 148-150). A summary of the previous studies on the recurrence risk of shoulder dystocia is presented below.

Baskett et al published in 1995 a study of 254 shoulder dystocia cases in 40 518 vaginal cephalic deliveries at Grace Maternity Hospital, Halifax, Canada (99). Of these 254 women who experienced shoulder dystocia, there was one woman who experienced recurrent shoulder dystocia. The author did not make any adjustment for potentially confounding factors.

Lewis et al published in 1995 a study of all vaginal deliveries during the period 1983-1992 at Louisiana State University Centre in Shreveport, Louisiana, USA (n=37 465) (148). Shoulder dystocia occurred in 747 cases (2%). Of these, 101 patients had 123 subsequent vaginal deliveries, and in 17 (13.8%) of these deliveries recurrent shoulder dystocia occurred. The authors did not make any adjustment for potentially confounding factors.

Ginsberg et al published in 2001 a study of all vaginal deliveries registered in the Northwestern University Medical Center obstetric database, Washington state, USA, (n=39 681) (150). There were 602 deliveries with shoulder dystocia (1.5%). Sixty-six of the women with shoulder dystocia had a subsequent vaginal delivery recorded at this hospital, of whom 11 (16.7%) had recurrent shoulder dystocia.

Mehta et al published in 2007 a study performed in Hutzel Women's Hospital in Detroit, Michigan, USA, during the period 1996-2000 (149). Of a total of 25 995 vaginal deliveries, 205 deliveries were affected by shoulder dystocia (0.8%). Forty-two of these women had a subsequent vaginal delivery in this hospital, of whom four experienced recurrent shoulder dystocia. The authors did not make any adjustment for potentially confounding factors. All of these studies suffered from limited sample sizes, and in addition a large proportion of the women were either lost to follow-up or had a subsequent cesarean delivery.

Moore et al published in 2008 a large population-based case-control study of deliveries in Washington State, USA, during the period 1987-2004 (44). The authors studied 26 208 vaginal deliveries with shoulder dystocia. Of these 7819 women underwent a subsequent vaginal delivery and 1060 experienced recurrent shoulder dystocia (13.5%). The overall occurrence of shoulder dystocia was as high as 2.3%. The relative impact of a prior shoulder dystocia, as compared to other risk factors of shoulder dystocia, was not addressed.

Knowledge of the recurrence rate of shoulder dystocia is important for deciding mode of delivery in a subsequent pregnancy. Knowledge of the risk factor for recurrence of shoulder dystocia is also important for patients counseling about the risk. In order to gain knowledge of the recurrence risks of a rare obstetric event, such as shoulder dystocia, large sample sizes are needed to ensure sufficient statistical power. It is also necessary to have information on other risk factories for shoulder dystocia so that adjustment can be made for possible confounding factors.

Background for Paper II

Parity and the risk of shoulder dystocia

To our knowledge, the association of parity with shoulder dystocia has been addressed in three previous studies, all of them suggesting no association of parity with the risk of shoulder dystocia (33, 151, 152). However, during our work with Paper I in this thesis, we found surprisingly that shoulder dystocia occurred more often in women with a previous delivery than in first time mothers (153). A summary of the three previous studies on the association of parity and the risk of shoulder dystocia is presented below.

Nocon et al published in 1993 a retrospective study of 12 532 vaginal deliveries during the period 1986-1990 at the Wishard Memorial Hospital, Indianapolis, Indiana, USA, and assessed the risk of shoulder dystocia according to various risk factors (33). The authors suggested that parity (para >0), among other variables, was not associated with the risk of shoulder dystocia.

Mocanu et al published in 2000 the outcome of all deliveries of offspring weighing 4500 grams or more (n=828) at the Coombe Women's Hospital in Dublin, Ireland during the period 1991-1995 (151). Parity (para 0 versus para >0) was one among many study factors. They found that the occurrence of shoulder dystocia was similar in primiparous and parous women. No adjustments were made for potentially confounding factors.

Revicky et al published in 2011 a retrospective cohort study of 9767 deliveries (234 shoulder dystocia cases) in the Norfolk and Norwich University Hospital, UK during the period 2005-2007 (152). They studied the association of various study factors, including parity (para 0 versus para >0) with shoulder dystocia. After adjustment for other study factors, the authors concluded that parity was not an independent risk factor for shoulder dystocia.

Few studies on the association of parity with shoulder dystocia have had the power to adjust for possible confounding factors. Also, type II statistical errors may occur, due to low statistical power in the studies. In addition, as far as we know, no study has investigated the association of birthweight with shoulder dystocia within strata of parity. Thus, better studies are needed to determine whether parity is an independent risk factor for shoulder dystocia.

Background for Paper III

Pregnancy week at delivery and the risk of shoulder dystocia

It has been suggested that giving birth at pregnancy week 42 or later, regarded as postterm pregnancy, may increase the risk of shoulder dystocia (6). Offspring birthweight increases by increasing pregnancy weeks, and for that reason alone shoulder dystocia may occur more often in postterm deliveries. Nonetheless, it is uncertain whether postterm delivery is an independent risk factor for shoulder dystocia. A summary of previous studies on pregnancy week at delivery and the risk of shoulder dystocia is presented below.

Hopwood published in 1982 a study of 17 735 deliveries (92 shoulder dystocia cases) at the Arlington Community Hospital, Virginia, USA (11). The author noted that 10% of the deliveries were postterm, but 27% of the shoulder dystocia cases were postterm. This observation suggested postterm pregnancy as a risk factor of shoulder dystocia. Adjustment for birthweight was not made.

Acker et al published in 1985 a study of vaginal deliveries of offspring weighing 2500 grams or more, (n=14 721; 309 with shoulder dystocia) at Beth Israel Hospital, Boston, Massachusetts, USA (31). The study population was divided into birthweight categories of 500 grams. The authors found a 70% higher risk for shoulder dystocia in postterm compared to term deliveries for offspring weighing >4500 grams (RR 1.7). Acker et al states: *“The number of cases of shoulder dystocia that occurred in postterm deliveries of still lighter offspring was not great, but the frequency were consistently (but not significantly) larger than in term deliveries.”* This study is used as a reference for the American College of Obstetrics and Gynecology when listing postterm pregnancy as one of the risk factors for shoulder dystocia (6).

Baskett et al published in 1995 a study of 254 shoulder dystocia cases in 40 518 vaginal cephalic deliveries at Grace Maternity Hospital, Halifax, Canada (99). They found that the occurrence of shoulder dystocia was increased three-fold in postterm deliveries (42 weeks or beyond). Adjustments for birthweight or other potential confounding factors was not made.

Campbell et al published in 1997 a study of term (n=379 445) and postterm (n=65 796) deliveries in Norway (154). Within strata of birthweight the authors found no higher risk of shoulder dystocia in postterm deliveries as compared to term deliveries.

Two of the above studies were descriptive studies only (11, 99). The other two did analyses within strata of birthweight. No adjustments for other potential confounding factors were made in any of the studies (31, 154). Also, the results are conflicting. Therefore there is a need for studies which have the statistical power to adjust for potential confounding factors. None of the previous studies had any information on maternal diabetes according to pregnancy week at delivery and the risk of shoulder dystocia. It is therefore not known whether women with diabetes are at particularly high risk of shoulder dystocia at pregnancy week 40 or beyond. Studies that can answer this question are therefore needed.

Previous studies have lacked the statistical power to draw conclusions about the recurrent risk of shoulder dystocia. Also, previous studies have shown conflicting results regarding the association of parity with the risk of shoulder dystocia. Furthermore, there is insufficient knowledge on pregnancy week at delivery and the risk of shoulder dystocia.

2.0 AIMS

Aims of Paper 1: To estimate the absolute risk and the relative risk of shoulder dystocia in the second delivery according to history of shoulder dystocia and offspring birthweight.

Aims of Paper 2: To estimate the association of parity and offspring birthweight with the risk of shoulder dystocia, and study whether the association of offspring birthweight with shoulder dystocia differs by parity.

Aims of Paper 3: To study whether pregnancy week at delivery is an independent risk factor for shoulder dystocia, and to study whether women with diabetes are at particularly high risk of shoulder dystocia at week 40 or later.

3.0 MATERIALS AND METHODS

3.1 Study populations

Our studies are population-based. All data were drawn from the Medical Birth Registry of Norway (MBRN). The MBRN is a national birth registry containing information on all deliveries in Norway. The MBRN was established in 1967, and was the first nation-wide birth registry in the world. The MBRN is based on compulsory notification of every delivery in Norway (155). The birth attendant in charge of the delivery is responsible for the reporting to the MBRN and all information about the mother, the pregnancy, the delivery and the newborn is reported shortly after the delivery by a standardized notification form. The original notification form was introduced at the start of the MBRN in 1967, and remained unchanged for 30 years (155). However, in December 1998, a revised and more comprehensive notification form was introduced.

More than 2.5 million births were registered by the end of 2009. From its inception, the MBRN has had a dual objective: surveillance of adverse outcomes and research (155).

Study population Paper I

We studied all women with two consecutive (first and second) deliveries of singletons in cephalic presentation after pregnancy week 16 in Norway during the period 1967-2005. In total, 593 144 women were included in the study. We then restricted the analyses to women who gave birth vaginally both the first and second time, which led to the exclusion of 38 373 women with a cesarean at first delivery, and additionally 17 457 women with a cesarean at second delivery. Selection of the study sample Paper I is shown in Figure 4.

First delivery

Cesarean delivery
n=38 373; 6.5%

Second delivery

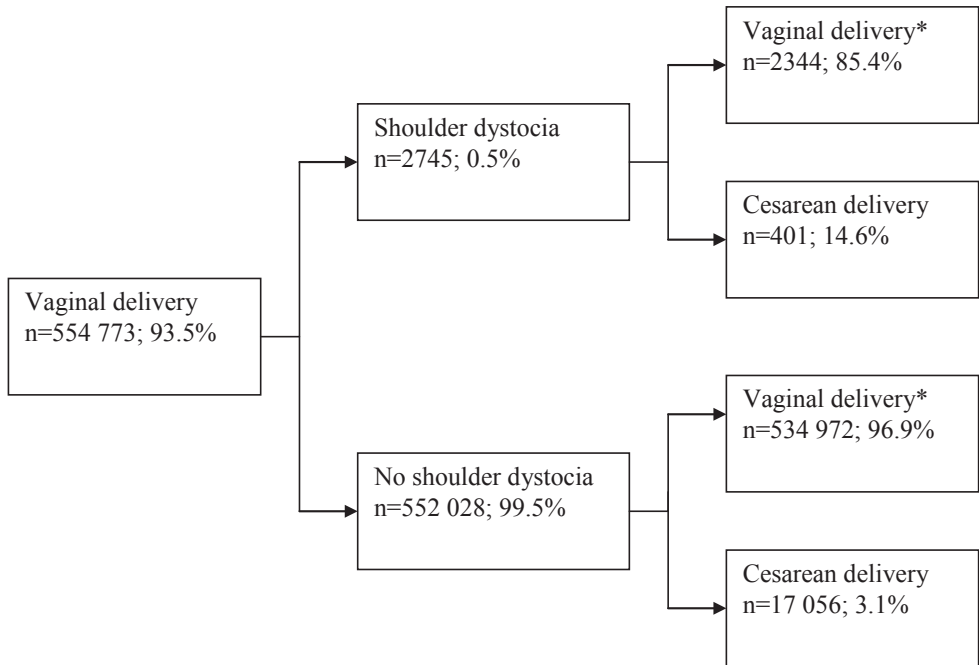


Figure 4. All women with one fetus in cephalic position according to mode of delivery in their first and second pregnancy in Norway during the period 1967-2005 (n= 593 144)

*Women included in our study sample.

Study population Paper II

We studied all deliveries after pregnancy week 16 in Norway during the period 1967-2006. The data were drawn from the MBRN and a total of 2 211 395 deliveries took place during our study period. We restricted the study to vaginal deliveries of singleton offspring in cephalic presentation. We therefore excluded 213 206 (9.6%) cesarean deliveries, 39 255 vaginal multiple births, 35 546 vaginal non-cephalic deliveries. Additionally we excluded 2513 deliveries with missing offspring birthweight. Thus 1 914 544 deliveries remained for the data analyses.

Study population Paper III

We studied all deliveries in Norway during the period 1967-2009 (n=2 521 086). The data were drawn from the MBRN. We restricted the study to vaginal deliveries of singleton offspring in cephalic presentation. We therefore excluded 67 024 multiple births, 85 996 non-cephalic deliveries, 190 178 (8.1%) cesarean deliveries and 7937 deliveries with missing information on presentation. Additionally we excluded 107 253 deliveries with missing information on pregnancy week at delivery and 2693 deliveries with missing information on birthweight. Since shoulder dystocia rarely occurs before pregnancy week 32 and an unknown number of shoulder dystocia cases occurring in week 44 or later may have been erroneously classified as such, we chose to restrict the study to deliveries between pregnancy weeks 32 and 43. We therefore excluded 19 042 deliveries before pregnancy week 32 and 26 006 deliveries at pregnancy week 44 or beyond. Thus a total of 2 014 956 deliveries remained for the data analyses.

3.2 Dependent variable

Shoulder dystocia

In Paper I the dependent variable was shoulder dystocia in the second delivery. In Papers II and III the dependent variable was shoulder dystocia at delivery. Information on shoulder dystocia was based on one item in the standardized MBRN notification form. The original notification form had an open space where delivery complications such as shoulder dystocia were reported. However, the revised notification form contains a specific item: "Vanskelig skulderforløsning" ("Difficult delivery of the shoulders"). There is a box to be ticked if the delivery of the shoulders was difficult. In our study shoulder dystocia at delivery was present if "difficult delivery of the shoulders" was reported to the MBRN.

3.3 Main independent variables

Paper I. Shoulder dystocia at first delivery

The main independent variable in Paper I was "shoulder dystocia at the first delivery", coded as "yes" or "no".

Paper II. Parity

The main independent variable in Paper II was parity. Parity was defined as the number of previous deliveries by the mother. We used the number of previous deliveries for categorization of parity: para 0, para 1 and para ≥ 2 .

Paper III. Pregnancy week at delivery

The main independent variable in Paper III was pregnancy week at delivery. In the MBRN, information on pregnancy week at delivery was given as completed number of pregnancy weeks. This is calculated from the term date which is estimated from information based on the mother's reported first day of last menstrual period. From 1999 the term date in the MBRN was estimated by ultrasonographic examination in pregnancy weeks 17-19 for 97% of the pregnancies. This examination is offered to all pregnant women free of charge. For the remaining 3% the term date was still estimated from information based on the mother's reported first day of last menstrual period. We categorized pregnancy week at delivery as: 32-35, 36-37, 38-39, 40-41 and 42-43 weeks.

3.4 Other study factors

Offspring birthweight

Offspring birthweight has been measured by the birth attendant shortly after delivery and reported in grams. In the data analyses we categorized birthweight as follows: <3000, 3000-3499, 3500-3999, 4000-4499, 4500-4999 and ≥ 5000 grams.

Induction of labor

This information is based on four variables in the MBRN: induction with prostaglandins (yes/no), induction with oxytocin (yes/no), induction with amniotomi (yes/no) and induction with other methods (yes/no). These four variables were joined into a new dichotomous variable: induction (yes/no). If one or more of these four original variables were coded as yes, they were given the value yes in the new variable.

Use of epidural analgesia

This is based on information from one variable in the MBRN: epidural analgesia (yes/no).

Prolonged labor

Defined in the MBRN as labor lasting more than 24 hours (yes/no).

Forceps-assisted delivery

Information in the MBRN is given as use of forceps (yes/no).

Vacuum-assisted delivery

Information in the MBRN is given as use of vacuum (yes/no).

Diabetes mellitus

In the MBRN the information on maternal diabetes is based on four variables: diabetes type 1, diabetes type 2, gestational diabetes or diabetes other types/unspecified diabetes (all yes/no). These four variables were joined and coded as diabetes (yes/no).

Maternal age

In the MBRN, maternal age is given as the mother's age (in years) at the time of delivery. In the data analyses we categorized maternal age as follows: <20, 20-24, 25-29, 30-34 and ≥35 years old.

Year (period) of delivery

This is based on the child's year of birth as reported to the MBRN. In the data analyses we categorized period of delivery as 1967-1976, 1977-1986, 1987-1996 and 1997-2005 in Paper I. In Paper II period of delivery was grouped as 1967-1976, 1977-1986, 1987-1996 and 1997-2006 and in Paper III the grouping was 1967-1976, 1977-1986, 1987-1996 and 1997-2009 .

3.5 Study design and statistical methods

Paper I

Design: Cohort study.

The proportion (%) of women who experienced shoulder dystocia in the first or in the second delivery was calculated. The risks of shoulder dystocia in the second delivery according to history of shoulder dystocia and other study factors were estimated as crude ORs (cOR) and adjusted ORs (aOR) with 95% CIs using logistic regression analyses. The absolute risks of shoulder dystocia in the second delivery as according to history of shoulder dystocia and offspring birthweight was calculated as proportions with 95% CIs. All statistical analyses were performed by using the Statistical Package for the Social Science (SPSS) for Windows version 14.0 (SPSS Inc., Chicago, Illinois, USA).

Paper II

Design: Cross-sectional study.

Proportions (%) of deliveries with shoulder dystocia were calculated across parity (0, 1 or more than 1 previous delivery), offspring birthweight and other study factors. Differences in proportions were tested using the chi-square test. The risks of shoulder dystocia

according to parity, offspring birthweight and other study factors were estimated as cOR and aOR with 95% CIs, using logistic regression analyses. These analyses were repeated in strata of parity (0, 1 or more than 1 previous delivery).

Possible interaction between birthweight and parity, associated with the risk of shoulder dystocia was explored by including an interaction term in the multivariate analyses, and the level of statistical significance was tested by applying the Wald test. All statistical analyses were performed using SPSS for Windows version 15.0 (SPSS Inc., Chicago, Illinois, USA).

Paper III

Design: Cross-sectional study.

We calculated the proportions (%) of deliveries with shoulder dystocia within the categories of pregnancy week at delivery in the study sample as a whole and within categories of offspring birthweight and other study factors. Differences in proportions were tested using the chi-square test. The relative risk of shoulder dystocia according to pregnancy week at delivery, offspring birthweight and other study factors were estimated as cOR and aOR with 95% CIs, using logistic regression analyses. The analyses were repeated for pregnancies with and without diabetes.

Possible interaction between pregnancy week at delivery and maternal diabetes, with the risk of shoulder dystocia was explored by including an interaction term in the multivariate analyses, and the level of statistical significance was tested by applying the Wald test. All statistical analyses were performed using the SPSS for Windows version 15.0 (SPSS Inc., Chicago, Illinois, USA).

3.6 Ethical considerations

Our studies were approved by the Publication Committee of the MBRN, and the MBRN is approved by The Norwegian Data Protection Authority (Datatilsynet). The study participants in the MBRN are anonymous to the researchers. According to the Norwegian Act of Health Research (Helseforskningsloven) §8, it is not necessary to apply for ethical approval to Regional Ethical Committees if the research is performed on anonymous data from the MBRN.

4.0 SYNOPSIS OF THE STUDIES

Paper I

Overland EA, Spydslaug A, Nielsen CS, Eskild A. Risk of shoulder dystocia in second delivery: does a history of shoulder dystocia matter? *American Journal of Obstetrics and Gynecology* 2009; 200:506.e1-6.

Objective: Our aim was to estimate the relative and absolute risk of shoulder dystocia in the second delivery according to history of shoulder dystocia and offspring birthweight.

Design: Population-based retrospective cohort study.

Setting: MBRN.

Population: All women with two consecutive (first and second) deliveries of singleton offspring in cephalic presentation during the period 1967-2005 (n=537 316).

Main outcome measure: Shoulder dystocia at the second delivery.

Results: In the second delivery, shoulder dystocia occurred in 0.8% of all women. In women with a prior shoulder dystocia the recurrence risk was 7.3%. Most shoulder dystocia cases in second delivery were in women without such history (96.2%). Offspring birthweight was the most important risk factor for shoulder dystocia in second delivery, cOR 292.9 (95% CI 237.8-360.7) comparing birthweight >5000 grams with 3000-3499 grams.

Conclusion: Prior shoulder dystocia increased the risk of shoulder dystocia in the second delivery. However, offspring birthweight was by far the most important risk factor.

Paper II

Øverland EA, Vatten LJ, Eskild A. Risk of shoulder dystocia: associations with parity and offspring birthweight. A population study of 1 914 544 deliveries. *Acta Obstetrica et Gynecologica Scandinavica* 2012; 91:483-488.

Objectives: We estimated the associations of parity and offspring birthweight with the risk of shoulder dystocia, and studied whether the association of offspring birthweight differed by parity.

Design: Population-based register study.

Setting: MBRN.

Population: All vaginal deliveries of a singleton offspring in cephalic presentation during the period 1967-2006 (n=1 914 544).

Main outcome measure: Shoulder dystocia at delivery.

Results: Shoulder dystocia occurred in 0.68% (13 109/1 914 544) of all deliveries. There was a strong positive association of birthweight with risk of shoulder dystocia, and 75% (9 765/13 109) of all cases occurred in deliveries of offspring weighing 4000 grams or more. The association of birthweight displayed similar patterns across parities, but the association was slightly stronger in parous than in primiparous women. Among first-time mothers, 0.12% (320/276 614) with offspring weighing 3000-3499 grams (reference) experienced shoulder dystocia, whereas 13.30% (169/1 244) with offspring birthweight higher than 5000 grams (OR 135.7, 95% CI 111.6-165.1) had shoulder dystocia. The corresponding results for women with one previous delivery were 0.08% (161/201 572) and 16.45% (501/3054) (OR 246.4, 95% CI 205.4-295.5).

Conclusions: High offspring birthweight is the major risk factor for shoulder dystocia and most cases occurred at high offspring birthweights. The positive association of birthweight with shoulder dystocia showed similar patterns across parities, but at high birthweights parous women were at greater risk of shoulder dystocia than primiparous women.

Paper III

Øverland EA, Vatten LJ, Eskild A. Pregnancy week at delivery and the risk of shoulder dystocia: a population study of 2 014 956 deliveries. *BJOG: An International Journal of Obstetrics & Gynecology* 2014; 121(1):34-42.

Objective: To study whether pregnancy week at delivery is an independent risk factor for shoulder dystocia.

Design: Population study.

Setting: MBRN.

Population: All vaginal deliveries of singleton offspring in cephalic presentation in Norway during the period 1967-2009 (n=2 014 956).

Methods: The incidence of shoulder dystocia was calculated according to pregnancy week at delivery. The associations of pregnancy week at delivery with shoulder dystocia were estimated as cORs and aORs using logistic regression analyses. We repeated the analyses in pregnancies with and without maternal diabetes.

Main outcome measures: Shoulder dystocia at delivery.

Results: The overall incidence of shoulder dystocia was 0.73% (n=14 820), and the incidence increased by increasing pregnancy week at delivery. Birthweight was strongly associated with shoulder dystocia. After adjustment for birthweight, induction of labor, use of epidural analgesia at delivery, prolonged labor, forceps- and vacuum-assisted delivery, parity, period of delivery and maternal age in multivariable analyses, the aOR for shoulder dystocia was 1.77 (95% CI 1.42-2.20) for deliveries at 32-35 weeks, and 0.84 (95% CI 0.79-0.88) at 42-43 weeks, using weeks 40-41 as the reference. In diabetic pregnancies (n=11 188), the incidence of shoulder dystocia was 3.95%, and after adjustment for birthweight the aOR for shoulder dystocia was 2.92 (95% CI 1.54-5.52) at weeks 32-35, and 0.91 (95% CI 0.50-1.66) at 42-43 weeks.

Conclusions: The risk of shoulder dystocia was associated with increased birthweight, maternal diabetes, induction of labor, use of epidural analgesia at delivery, prolonged labor, forceps- and vacuum-assisted delivery, parity and period of delivery, but not with postterm delivery.

5.0 DISCUSSION

5.1 Main findings

Paper I

- In the second delivery shoulder dystocia occurred in 0.8% of all women as compared to 0.5% of all women in the first delivery.
- A history of shoulder dystocia increased the risk of shoulder dystocia in the second delivery as compared to women without such a history, and 7.3% of women with a history of shoulder dystocia experienced a new shoulder dystocia.
- Very few of all shoulder dystocia cases in the second delivery occurred in women with a history of shoulder dystocia (3.8%).
- The combination of a history of shoulder dystocia and high offspring birthweight in the second pregnancy conferred a high risk of recurrence. If the birthweight was low or normal, the risk of shoulder dystocia in the second delivery was low, regardless of a history of shoulder dystocia.

Paper II

- High offspring birthweight was a major risk factor for shoulder dystocia and three quarters (75%) of all shoulder dystocia cases occurred in deliveries of offspring weighing more than 4000 grams.
- Parous women are at higher risk of shoulder dystocia compared to primiparous women, also after adjustment for offspring birthweight and other study factors.
- The positive association of birthweight with shoulder dystocia showed similar patterns across categories of parity, but at high birthweights parous women were at higher risk of shoulder dystocia than primiparous women.

Paper III

- The proportion of deliveries complicated with shoulder dystocia, increased by increasing pregnancy length at delivery.
- After adjustment for offspring birthweight there was a decreasing risk of shoulder dystocia with increasing pregnancy week. This finding was particularly pronounced in diabetic pregnancies.
- Consequently, postterm delivery is not an independent risk factor for shoulder dystocia.

5.2 Methodological considerations

Methodological considerations presented in the discussion sections of each paper included in this thesis will in general not be repeated in the following text.

Limitation of the findings

The aim of epidemiological studies is to produce an estimate which ideally is as close as possible to the true association between an exposure and an outcome. However, there will always be possible methodological limitations in the form of errors which may influence the measurements of association found in a study. In epidemiological studies there are two sorts of errors: *systematic errors and random errors* (109, 156).

Systematic errors, or biases, result in predictable, but often unknown fluctuations in measurement and are classified into *selection bias, information bias and confounding* (109, 157). *Random errors* exist independent of systematic errors, and creates unpredictable fluctuation of the variability in the data (157). Random error is affected by the size of the study. When the study size increases, random errors are reduced.

Selection bias

Selection bias in study inclusion may cause under- or overestimation of the prevalence of a given outcome. The participants in our studies were drawn from the MBRN, which includes all deliveries after pregnancy week 16 in Norway since 1967. Notification to the MBRN is compulsory, and it is unlikely that there are many, if any, deliveries that have not been reported. Hence, it is not likely that skewed selection to our studies has biased the estimated prevalence of shoulder dystocia.

In Paper I we followed all women in Norway with a first vaginal delivery to her next vaginal delivery. Women with a vaginal delivery at their first delivery, but a cesarean delivery at their second delivery were not included in the study population.

Women who experienced shoulder dystocia at their first delivery may have been more likely to have a cesarean delivery the second time than women without such a history. If this is true, we may have underestimated the recurrence of shoulder dystocia, since shoulder dystocia are not present in cesarean deliveries. Offspring birthweight showed the strongest association with our outcome. We calculated the mean birthweight among those

who experienced shoulder dystocia during the first delivery according to mode of delivery in the second delivery (cesarean delivery; yes/no). However, the mean birthweight was similar in the two groups (3940 grams and 3960 grams, respectively). Thus, it is not likely that selection to cesarean delivery in the second delivery according to offspring birthweight has biased the estimates of the association of history of shoulder dystocia with shoulder dystocia in the second delivery.

Information bias

Information bias occurs when the information on the exposure or the outcome is erroneous. This is often referred as misclassification. Misclassification can be *non-differential* or *differential*. Non-differential misclassification occurs when the misclassification is not systematically linked both to the outcome and the exposure. Generally, non-differential misclassification may render an underestimation of the association between an exposure and an outcome, thus biasing the results towards the null-hypotheses. However, although non-differential misclassification will “blur” the result and fade the possible associations, the direction of the associations is usually valid. It is generally assumed that non-differential misclassification occurs quite often in studies.

In contrast, differential misclassification occurs when the misclassification is systematically linked both to exposure and outcome. Differential misclassification may result in an erroneous estimate of the association between the exposure and the outcome.

Our outcome was shoulder dystocia at delivery. There is an agreement that shoulder dystocia is a situation where the fetal shoulders do not follow easily after the fetal head is born. Still there is a lack of uniform diagnostic criteria for shoulder dystocia and the diagnosis reminds to be based on the birth attendant’s subjective recognition. Therefore, an event that one birth attendant diagnoses as a shoulder dystocia, may not be diagnosed as such by another. It is therefore quite likely that misclassification of the outcome occurs in our study. This may affect the estimates of the occurrence of shoulder dystocia. If the reporting of shoulder dystocia also is linked to the exposure variable, the estimated associations may be erroneous.

In 1999 the reporting of shoulder dystocia to MBRN changed from reporting shoulder dystocia by an open space of any complication at delivery (“Komplikasjoner i forbindelse med fødsel; Nei/ Ja (spesifiser)”) to ticking a box in response to a direct question on shoulder dystocia (Figure 5 and Figure 6). This change in reporting might have affected the reported occurrence of shoulder dystocia, which increased from 0.9% in 1998 to 1.3% in 1999. Year of delivery (period) was included to adjust for changes of reporting over time.

Registreringsskjema fra 1967-1998

STATENS HELSETILSYN
Postboks 8128 Dep.
0032 OSLO

Medisinsk registrering av fødsel

Sendes 9. dag etter fødselen til
fylkeslegen (stadstyskerus) i det
fylket der moren er bosatt.

Merk: Det skal fylles ut blankett for hvert barn (foster). Der barnet etter fødselen, skal det også fylles ut legeerklæring om
dødsfall, og/eller dødsfallet meldes til skifteretten (lensmannen).


Barnet	Barnet var 1 <input type="checkbox"/> Levende født 2 <input type="checkbox"/> Dødfødt foster	Født dag, mnd., år	Klokkeslett	Persønnr.	Skriv ikke her	
	1 <input type="checkbox"/> Enkel 2 <input type="checkbox"/> Tvilling 3 <input type="checkbox"/> Trilling 4 <input type="checkbox"/> Firling	Kjønn 1 <input type="checkbox"/> Gutt 2 <input type="checkbox"/> Pika				
	Etternavn, alle fornavn (bare for levendefødte)					
	Fødested. Navn og adresse på sykehuset/fødestedet					
Faren	Etternavn, alle fornavn		Født dag, mnd., år	Bostedekommune		
Moren	Etternavn, alle fornavn. Pikenavn		Født dag, mnd., år			
	Bosted. Adresse		Kommune			
	Ektekapselig status 1 <input type="checkbox"/> Ugilt 6 <input type="checkbox"/> Samboende 2 <input type="checkbox"/> Gilt 3 <input type="checkbox"/> Enke 4 <input type="checkbox"/> Separert 5 <input type="checkbox"/> Skilt		Ektekapsår (gilde)			
	Antall tidligere fødte (for denne fødselen)		Levende fødte	Av disse i live	Dødfødte	
	Er moren i slett med faren? 1 <input type="checkbox"/> Nei 2 <input type="checkbox"/> Ja. Hvilket slettskapsforhold:					
Morens helse før svangerskapet	1 <input type="checkbox"/> Normal 2 <input type="checkbox"/> Sykdom (spesifiser):		Siste menstruasjons første blødningsdag			
Morens helse under svangerskapet	1 <input type="checkbox"/> Normal 2 <input type="checkbox"/> Komplikasjoner (spesifiser):					
Ble fødselen provosert	1 <input type="checkbox"/> Nei 2 <input type="checkbox"/> Ja					
Inngrep under fødselen	1 <input type="checkbox"/> Nei 2 <input type="checkbox"/> Ja (spesifiser): Inngrepet utført av 1 <input type="checkbox"/> Lege 2 <input type="checkbox"/> Jordmor					
Komplikasjoner i forbindelse med fødselen	1 <input type="checkbox"/> Nei 2 <input type="checkbox"/> Ja (spesifiser):					
Føtalvann, placenta og navlesnor	1 <input type="checkbox"/> Normalt 2 <input type="checkbox"/> Patologisk (spesifiser):					
Barnets tilstand	Bare for levende fødte. Tegn på asfykasi?		Apgarscore etter 1 min.	etter 5 min.		
	1 <input type="checkbox"/> Nei 2 <input type="checkbox"/> Ja					
	For levende fødte og dødfødte. Tegn på medfødt anomali, på skade eller sykdom?					
	1 <input type="checkbox"/> Nei 2 <input type="checkbox"/> Ja. Hvilke:					
Barnets tilstand	Lengde (i cm)	Hode-omkr. (i cm)	Vekt (i g)	For døde innen 24 timer Livet varte i	Timer Min	
	For dødfødte. Døden inntrådte 1 <input type="checkbox"/> Før fødselen 2 <input type="checkbox"/> Under fødselen					
	Dødsårsak					
Avbrutte arvelige lidelser i slekten	Seksjon? 1 <input type="checkbox"/> Nei 2 <input type="checkbox"/> Ja					
	1 <input type="checkbox"/> Nei 2 <input type="checkbox"/> Ja Sykdommens art og hos hvilke slektninger:					

50 000.5.96.334 GRAVTE


Sted (sykehusets stempel) Dato Jordmor Lege

IK - 1002.

Figure 5. The standardized notification form of the Medical Birth Registry of Norway used from 1967 throughout 1998



Melding om avsluttet svangerskap etter 12. uke – Fødsel, dødfødsel, spontanabort
Se utfyllingsinstruks for blanketten på bakgrunnen



Sosial- og helsedirektoratet

A	Institusjonsnr:	Institusjonsnavn:		Fødsel utenfor institusjon:	Mors fulle navn og adresse:
	Mors sivilstatus:	<input type="checkbox"/> Gift <input type="checkbox"/> Ugift/erslig <input type="checkbox"/> Annet <input type="checkbox"/> Samboer <input type="checkbox"/> Skilt/separert/enke		<input type="checkbox"/> Hjemme, planlagt <input type="checkbox"/> Hjemme, ikke planlagt <input type="checkbox"/> Under transport <input type="checkbox"/> Annet sted	Pikenavn (etternavn):
	Slektskap mellom barnets foreldre?	<input type="checkbox"/> Nei <input type="checkbox"/> Hvis ja, hvorledes:		Mors bosted:	
	Fars fødselsdato:	Fars fulle navn:		Mors fødselsnr.:	
B	Siste menstr. t. blødn. dag:	<input type="checkbox"/> Sikker <input type="checkbox"/> Usikker <input type="checkbox"/> Mors tidligere svangerskapsfødsel		Levende-fødsel Dødfødsel (24 uke og over) Spontanabort/Dødfødsel (12–23 uke) Spontanaborter (under 12 uke)	
	Ultralyd utført?	<input type="checkbox"/> Nei <input type="checkbox"/> Ja, UL termin:		Annet prenatal diagnostikk? <input type="checkbox"/> Nei <input type="checkbox"/> Ja, angi type:	Patologiske funn ved prenatal diagnostikk? <input type="checkbox"/> Nei <input type="checkbox"/> Ja, hvis bekreftet – spesifiser
B	Spesielle forhold for svangerskapet:	<input type="checkbox"/> Intet spesielt <input type="checkbox"/> Allergi <input type="checkbox"/> Astma <input type="checkbox"/> Kronisk ryngesykdom <input type="checkbox"/> Epilepsi <input type="checkbox"/> Tidligere sectio <input type="checkbox"/> Kronisk hypertensjon <input type="checkbox"/> Diabetes type 1 <input type="checkbox"/> Diabetes type 2 <input type="checkbox"/> Res. urinveisinfeksjon <input type="checkbox"/> Reumatoid artritt <input type="checkbox"/> Hjertesykdom <input type="checkbox"/> Annet, spesifiser i «B»:		Regelmessig kosttilskudd: <input type="checkbox"/> Nei <input type="checkbox"/> Ja, for sv. sk. 1 sv. sk. Multivitaminer <input type="checkbox"/> Folsyrefolsyre	B
	Spesielle forhold under svangerskapet:	<input type="checkbox"/> Intet spesielt <input type="checkbox"/> Blødning < 13 uke <input type="checkbox"/> Hypertensjon alene <input type="checkbox"/> Eklamsi <input type="checkbox"/> Blødning 13–28 uke <input type="checkbox"/> Preeklamsi lett <input type="checkbox"/> Hb < 9.0 g/dl <input type="checkbox"/> Blødning > 28 uke <input type="checkbox"/> Preeklamsi alvorlig <input type="checkbox"/> Hb > 13.5 g/dl <input type="checkbox"/> Glukosuri <input type="checkbox"/> Preeklamsi før 34. uke <input type="checkbox"/> Trombose, beh. <input type="checkbox"/> Svangerskapsdiabetes <input type="checkbox"/> HELLP syndrom <input type="checkbox"/> Infeksjon, spes. i «B»		Legemidler i svangerskapet: <input type="checkbox"/> Nei <input type="checkbox"/> Ja – spesifiser i «B»	
C	Røyking og yrke	Røyker mor ved sv. sk. begynnelse? <input type="checkbox"/> Nei <input type="checkbox"/> Daglig <input type="checkbox"/> Av og til Ant. sig. dagl.: - ved sv. sk. avslutning? <input type="checkbox"/> Nei <input type="checkbox"/> Daglig <input type="checkbox"/> Av og til Ant. sig. dagl.:		Mors yrke: <input type="checkbox"/> Samtykker ikke for yrkesoppd. <input type="checkbox"/> Ikke yrkesaktiv <input type="checkbox"/> Yrkesaktiv heltd. <input type="checkbox"/> Yrkesaktiv deltd.	Mors yrke:
	Leie/presentasjon:	<input type="checkbox"/> Sele <input type="checkbox"/> Normal bakthode <input type="checkbox"/> Tverrløse <input type="checkbox"/> Anvikende dødfødsel <input type="checkbox"/> Sectio <input type="checkbox"/> Annet, spesifiser i «C»:		Fødselstart: <input type="checkbox"/> Spontan <input type="checkbox"/> Sectio Ev. induksjonsmetode: <input type="checkbox"/> Prostoglandin <input type="checkbox"/> Oxytoben <input type="checkbox"/> Amniotomi <input type="checkbox"/> Annet, spesifiser i «C»:	Indikasjon for inngrep og/eller induksjon: <input type="checkbox"/> Komplikasjoner som beskrevet nedenfor <input type="checkbox"/> Fostermisfannelser <input type="checkbox"/> Overlid <input type="checkbox"/> Annet, spesifiser i «C»:
C	Inngreptilfalle:	<input type="checkbox"/> Ingen <input type="checkbox"/> Utskj. tang, hodeleie <input type="checkbox"/> Fremh. ved selvfødsel: <input type="checkbox"/> Vanlig fremhjelp <input type="checkbox"/> Sectio: <input type="checkbox"/> Var sectio planlagt før fødsel? <input type="checkbox"/> Nei <input type="checkbox"/> Ja <input type="checkbox"/> Vakuumekstraktor <input type="checkbox"/> Ultratrakning <input type="checkbox"/> Utført som elekt. sectio <input type="checkbox"/> Episiotomi <input type="checkbox"/> Tang på etterk. hode <input type="checkbox"/> Utført som akut. sectio		Komplikasjoner: <input type="checkbox"/> Ingen <input type="checkbox"/> Vannang, 12–24 timer <input type="checkbox"/> Placenta previa <input type="checkbox"/> Blødn. > 1500 ml, transf. <input type="checkbox"/> Truende intrauterin astyksi <input type="checkbox"/> Vannang > 24 timer <input type="checkbox"/> Abruptio placenta <input type="checkbox"/> Blødning 500–1500 ml <input type="checkbox"/> Risvekkelse, stimuleri <input type="checkbox"/> Mekaniske misforhold <input type="checkbox"/> Pervicinitet (grad 1-2) <input type="checkbox"/> Eklamsi under fødsel <input type="checkbox"/> Langsom fremgang <input type="checkbox"/> Vanskkelig skulderforløsning <input type="checkbox"/> Spindelmultipur (gr. 3-4) <input type="checkbox"/> Navlesnorforf. <input type="checkbox"/> Uterus atoni <input type="checkbox"/> Annet	C
	Anestesi/analgesi:	<input type="checkbox"/> Ingen <input type="checkbox"/> Lysgass <input type="checkbox"/> Epidural <input type="checkbox"/> Pudendal <input type="checkbox"/> Paraocervical blokk <input type="checkbox"/> Pelidin <input type="checkbox"/> Spinal <input type="checkbox"/> Infusjon <input type="checkbox"/> Narkose <input type="checkbox"/> Annet			
D	Placenta:	<input type="checkbox"/> Normal <input type="checkbox"/> Koagler <input type="checkbox"/> Navlesnor <input type="checkbox"/> Normal <input type="checkbox"/> Annet omslyng <input type="checkbox"/> Fostervann <input type="checkbox"/> Hinnerester <input type="checkbox"/> Utskrapping <input type="checkbox"/> Velantest feste <input type="checkbox"/> Ekte knute <input type="checkbox"/> Polyhydramnion <input type="checkbox"/> Misfarget <input type="checkbox"/> Ufullstendig <input type="checkbox"/> Marginal feste <input type="checkbox"/> Navlesnor- <input type="checkbox"/> Ekte knute <input type="checkbox"/> Oligohydramnion <input type="checkbox"/> Strikende, infisert <input type="checkbox"/> Infarkter <input type="checkbox"/> Placenta- <input type="checkbox"/> Karanomali <input type="checkbox"/> Navlesnor- <input type="checkbox"/> Ekte knute <input type="checkbox"/> Oligohydramnion <input type="checkbox"/> Blodtilblandet		Komplikasjoner hos mor etter fødsel: <input type="checkbox"/> Intet spesielt <input type="checkbox"/> Mor overflyttet <input type="checkbox"/> Feber > 38.5° <input type="checkbox"/> Mor intensivbeh. <input type="checkbox"/> Trombose <input type="checkbox"/> Sepsis <input type="checkbox"/> Eklamsi post partum <input type="checkbox"/> Annet, spesifiser	
	Fødselsdato:	Klokken:	Pluralitet: <input type="checkbox"/> Enkeltfødsel <input type="checkbox"/> For flerfødsel: <input type="checkbox"/> Nr. <input type="checkbox"/> Av totalt Kjønn: <input type="checkbox"/> Gutt <input type="checkbox"/> Pige <input type="checkbox"/> Barnets vekt: <input type="checkbox"/> Hode- <input type="checkbox"/> Total lengde: <input type="checkbox"/> Apper score: 1 min Ved liv spesifiser i «D»: <input type="checkbox"/> Ved dødfødsel: <input type="checkbox"/> Usikkert kjønn <input type="checkbox"/> Hode- <input type="checkbox"/> Eventuelt sele- <input type="checkbox"/> 5 min		
D	Barnet var:	For dødfødsel: <input type="checkbox"/> Dødfødsel <input type="checkbox"/> Død før fødsel <input type="checkbox"/> Død under fødselen <input type="checkbox"/> Død etter innkomst For dødfødsel, oppg. også: <input type="checkbox"/> Dødfødsel <input type="checkbox"/> Død før innkomst <input type="checkbox"/> Død etter innkomst		Levendefødt, død innen 24 timer: <input type="checkbox"/> Livet varle <input type="checkbox"/> Timer <input type="checkbox"/> Min Dødsdato: <input type="checkbox"/> Dødsdato: <input type="checkbox"/> Dødsdato:	Klokken: <input type="checkbox"/> Klokken: <input type="checkbox"/> Klokken:
	Overført, barneavd.:	<input type="checkbox"/> Nei <input type="checkbox"/> Ja <input type="checkbox"/> Nei <input type="checkbox"/> Ja <input type="checkbox"/> Nei <input type="checkbox"/> Ja		Indikasjon for overflytting: <input type="checkbox"/> Respirasjonsproblem <input type="checkbox"/> Medfødte misd. <input type="checkbox"/> Annet, spesifiser <input type="checkbox"/> Prematur <input type="checkbox"/> Perinatale infeksjoner	Behandlingsstoder: <input type="checkbox"/> Lysbehandling <input type="checkbox"/> Lysbehandling <input type="checkbox"/> Systemisk antibiotika <input type="checkbox"/> Utskifting <input type="checkbox"/> Utskifting <input type="checkbox"/> Respiratorbehold. <input type="checkbox"/> CPAP beh. <input type="checkbox"/> Årsak:
D	Neonatale diagn.:	<input type="checkbox"/> Hypoglyk. (< 2 mmol/l) <input type="checkbox"/> Transf. tachypnoe <input type="checkbox"/> Cerebral infusjon <input type="checkbox"/> Korunktilt. beh. <input type="checkbox"/> Med. anemi (Hb < 13.5 g/dl) <input type="checkbox"/> Resp. distress syndr. <input type="checkbox"/> Cerebral depresjon <input type="checkbox"/> Navle/hudinf. beh. <input type="checkbox"/> Høtteledsdispl. beh. m. p. <input type="checkbox"/> Aspirasjonsyndrom <input type="checkbox"/> Abstinens <input type="checkbox"/> Perinat. inf. bakterielle <input type="checkbox"/> Facialsparase <input type="checkbox"/> Intet spesielt <input type="checkbox"/> Intrakraniell blødning <input type="checkbox"/> Neonatale krampes <input type="checkbox"/> Perinat. inf. andre <input type="checkbox"/> Plexusskade		Tegn til medfødte misdannelser: <input type="checkbox"/> Nei <input type="checkbox"/> Ja <input type="checkbox"/> Nei <input type="checkbox"/> Ja <input type="checkbox"/> Nei <input type="checkbox"/> Ja	Årsak: <input type="checkbox"/> ABO uforlik <input type="checkbox"/> RH immunisering <input type="checkbox"/> Fysiologisk <input type="checkbox"/> Annen årsak
	Tegn til medfødte misdannelser:	<input type="checkbox"/> Nei <input type="checkbox"/> Ja <input type="checkbox"/> Nei <input type="checkbox"/> Ja <input type="checkbox"/> Nei <input type="checkbox"/> Ja		Utskrivningsdato: <input type="checkbox"/> Utskrivningsdato:	

Protokollnr.: /

Lege: _____

Jordmor vifødsel: _____

Jordmor vutskrivning: _____

Lege barse/barnesv. _____

Barn: _____

Figure 6. The standardized notification form of the Medical Birth Registry of Norway introduced in December 1998

In Paper I we studied the recurrence risk of shoulder dystocia. The outcome variable was shoulder dystocia in the second delivery, whereas the exposure variable was shoulder dystocia in the first. If the likelihood of being diagnosed with shoulder dystocia in the second delivery was affected by the mother's history of shoulder dystocia, the estimated risk of recurrence may have been overestimated. On the contrary, birth attendants at deliveries of women with a history of shoulder dystocia, may make preparations that possibly prevent shoulder dystocia. In such case, the recurrences risk may be underestimated.

In Paper II the outcome variable was shoulder dystocia and the main exposure variables were parity and birthweight. Being parous is assumed to protect against delivery complications, therefore parous women may be less likely to be diagnosed with shoulder dystocia. If this is the case, the estimated risk of shoulder dystocia among parous women in our study, may represent an underestimate. It is also possible that the likelihood of being diagnosed with shoulder dystocia increased by increasing offspring birthweight. If this is the case, the positive association we found between birthweight and risk of shoulder dystocia may represent an overestimate.

In Paper III the main exposure variable was pregnancy week at delivery. It is often assumed that being postterm is an independent risk factor for shoulder dystocia. It is therefore possible that the likelihood of being diagnosed with shoulder dystocia increases by length of pregnancy. If this is the case, the inverse association we found between pregnancy week at delivery and risk of shoulder dystocia may represent an underestimate. Maternal diabetes is a known risk factor for shoulder dystocia. Therefore, mothers with diabetes may have increased likelihood of being diagnosed with shoulder dystocia. We found, however, an inverse association of pregnancy week and risk of shoulder dystocia both in pregnancies with and pregnancies without diabetes.

It is also possible that the diagnosis of shoulder dystocia in our papers is more often given to mothers with presence of the other study factors: epidural analgesia, operative delivery, prolonged labor and high maternal age. Such deliveries are often regarded as high-risk deliveries and draw special attention. This may result in a more liberal use of the shoulder dystocia diagnosis and the association between these study factors and the risk of shoulder

dystocia may have been overestimated. On the other hand, these high-risk deliveries may have drawn special attention so the birth attendants have done preparations that possibly prevent shoulder dystocia. If this is the case, the estimated associations may have been underestimated.

Confounding

Confounding may have biased our associations if there are underlying factors that are associated with both shoulder dystocia at delivery and one or more of the exposure variables, and these factors are not accounted for in the data analyses. Based on knowledge from previous studies birthweight is an important risk factor for shoulder dystocia. In our studies, offspring birthweight was a potential confounding factor since birthweight is associated both with the various exposure variables (previous shoulder dystocia, parity and gestational length) and outcome (shoulder dystocia). Thus in all three studies, offspring birthweight was included in the multivariate statistical models as a potential confounding factor. We also included the following potential confounding factors: maternal diabetes, induction of labor, epidural analgesia, prolonged labor, vacuum- and forceps-assisted delivery, maternal age. However, it is possible that potential confounding remains.

Obesity is associated with birthweight and possibly with shoulder dystocia. We lacked information on maternal weight and height since this information were not available in the MBRN during our study periods. Thus, we could not make adjustment for maternal obesity calculated as body mass index. However, results from previous studies do not strongly suggest that maternal body mass index nor maternal weight gain during pregnancy are independent risk factors for shoulder dystocia. Thus, these factors are not likely to be important confounders in our studies.

Interaction

Interaction exists when the effect of one exposure variable on the outcome is different in different exposure groups, for example groups of women with different parity (109, 157). Separate data analyses within groups (stratification) may be used to identify interaction. The strength of the interaction may be assessed by including an interaction term in multivariate model. In Paper II we stratified the study population into categories of parity (Para 0, Para 1 and Para ≥ 2) and studied whether the association of birthweight with shoulder dystocia differed by parity. We found a slightly stronger association among

parous mothers than among primiparous mothers. The strength of the interaction was assessed by including an interaction term (birthweight * parity) in the multivariate model, and we found a significant interaction.

In Paper III we stratified the population by the diabetes status of the mother and estimated the association of pregnancy week at delivery with shoulder dystocia in both women with diabetes and women without diabetes. We found a stronger association among women with diabetes than among women without diabetes. We assessed the strength of the interaction by including an interaction term (pregnancy week at delivery * maternal diabetes) in the multivariate model and found evidence of a significant interaction between pregnancy week at delivery and maternal diabetes regarding the risk of shoulder dystocia (Figure 5).

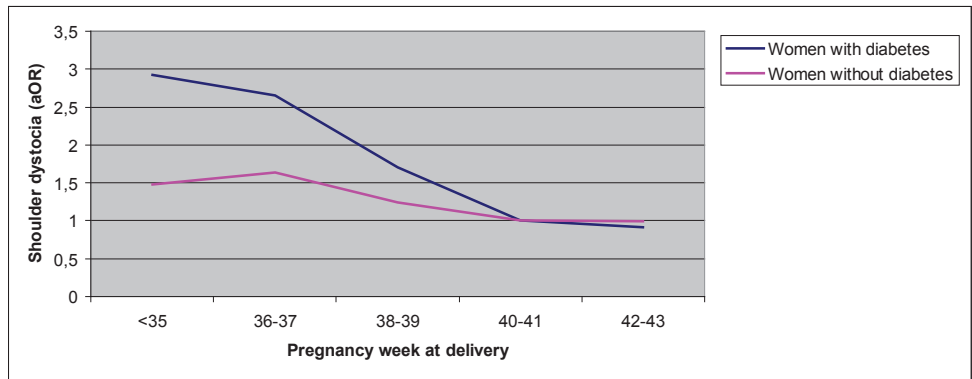


Figure 7. Adjusted odds ratios of shoulder dystocia according to pregnancy week at delivery among women with diabetes and women without diabetes. Adjustment made for offspring birthweight

6.0 CLINICAL IMPLICATIONS OF FINDINGS

Paper I

Among women who had shoulder dystocia in the first delivery, 7.3% had recurrent shoulder dystocia in the second delivery. There was a strong association between birthweight and the risk of shoulder dystocia. The combination of a history of shoulder dystocia and high offspring birthweight (≥ 4500 grams) in the second delivery yielded a high risk of recurrence. In contrast, when offspring birthweight was normal or lower, the risk of recurrence was low (Table 3).

Table 3. Absolute risk (%) with 95% CI for shoulder dystocia at second delivery among women with and without shoulder dystocia at first delivery according to birthweight

	Shoulder dystocia at first delivery (%)	
	Yes (95%CI)	No (95%CI)
Birthweight (grams)		
<3000	1.4 (0.1-4.0)	0.0 (0.0-0.0)
3000-3499	0.3 (-0.3-0.9)	0.1 (0.1-0.1)
3500-3999	2.2 (1.3-3.2)	0.4 (0.3-0.4)
4000-4499	9.7 (7.5-11.8)	1.8 (1.7-1.9)
4500-4999	19.9 (15.4-24.3)	6.3 (6.0-6.6)
>5000	29.2 (16.3-42.0)	17.4 (15.9-18.9)
Total	7.2 (6.2-8.3)	0.8 (0.08-0.08)

Our findings suggest that the assumption “once a shoulder dystocia always a cesarean” should be modified. When counseling women with a history of shoulder dystocia, clinicians must adopt an individual approach. A vaginal delivery can be recommended to a woman with a history of shoulder dystocia if she is expecting an offspring with a low to normal estimated birthweight, because her risk of shoulder dystocia is not higher than the risk among a general population of first time mothers. On the other hand, a planned cesarean delivery is an appropriate recommendation for a woman with a history of shoulder dystocia who is expecting an offspring with estimated high offspring birthweight (≥ 4500 grams).

Paper II

Parous women had a higher overall risk of shoulder dystocia compared to first-time mothers.

However, the increased risk among parous women was only present in deliveries of high birthweight offspring. To illustrate: Among women carrying an offspring weighing more than 5000 grams, a primiparous women had a 13.6% risk of experiencing shoulder dystocia at delivery, whereas a woman who has given birth once before had a 16.5% risk. At lower birthweights, the overall risk of shoulder dystocia was low and appeared to be lowest for parous women (Table 4).

Table 4. Absolute (%) risk with 95% CI for shoulder dystocia according to parity and offspring birthweight

	Shoulder dystocia (%)		
	Para 0 (95%CI)	Para 1 (95%CI)	Para ≥ 2 (95%CI)
Birthweight (grams)			
<3000	0.0 (0.0-0.0)	0.0 (0.0-0.0)	0.0 (0.0-0.0)
3000-3499	0.1 (0.1-1.1)	0.1 (0.1-0.1)	0.1 (0.0-0.1)
3500-3999	0.5 (0.4-0.5)	0.4 (0.3-0.4)	0.3 (0.3-0.3)
4000-4499	1.8 (1.7-1.9)	1.8 (1.7-1.9)	1.5 (1.4-1.6)
4500-4999	6.0 (5.6-6.4)	6.3 (6.0-6.6)	4.9 (4.6-5.2)
>5000	13.6 (12.6-14.6)	16.5 (15.1-17.8)	14.6 (13.4-15.8)
Total	0.6 (0.4-0.7)	0.8 (0.7-0.9)	0.8 (0.6-0.9)

Three out of four cases of shoulder dystocia occurred in deliveries of offspring weighing more than 4000 grams. Parous women represented 60% of the total number of the deliveries, but 67% of all cases of shoulder dystocia. Thus, doctors and midwives should be aware that high offspring birthweight is by far the most important risk factor for shoulder dystocia, and that multiparity is not a protective factor.

Paper III

The proportions of deliveries with shoulder dystocia increased with increasing pregnancy week at delivery. However, after adjustment for birthweight the direction of the association was reversed, and we found a decreasing risk of shoulder dystocia with increasing pregnancy week at delivery. Therefore, our findings suggest that postterm pregnancy is not an independent risk factor for shoulder dystocia (Table 5).

Table 5. Crude and adjusted ORs with 95% CI for shoulder dystocia according to pregnancy week at delivery

	Crude OR (95% CI)	Adjusted OR (95% CI)
Pregnancy week at delivery		
32-35	0.27 (0.22-0.33)	1.68 (1.35-2.10)
36-37	0.50 (0.45-0.55)	1.92 (1.74-2.12)
38-39	0.69 (0.66-0.73)	1.29 (1.24-1.34)
40-41	1.0	1.0
42-43	1.17 (1.11-1.22)	0.88 (0.84-0.92)

Women with diabetes were at especially high risk for shoulder dystocia. In our study, shoulder dystocia occurred in 0.73% of all deliveries, but the occurrence was more than five-fold higher among women with diabetes (3.95%).

Our findings suggest that at a given birthweight delivery before term is associated with a higher risk of shoulder dystocia than delivery at term or postterm. To illustrate this we have made additional analyses. In women without diabetes, the absolute risk of shoulder dystocia was 13.6% when giving birth to a high birthweight offspring (≥ 4500 grams) in week 37 as compared to 6.2% giving birth to an offspring of the same weight in week 42 (Table 6). Among women with diabetes expecting a high birthweight offspring (≥ 4500 grams), the absolute risk of shoulder dystocia was generally high, and above 17%, regardless of pregnancy week of delivery (Table 6).

Table 6. Absolute risk (%) of shoulder dystocia among women without or with diabetes expecting a high offspring birthweight (≥ 4500 grams) according to pregnancy week at delivery

Pregnancy week at delivery	Shoulder dystocia			
	Women without diabetes		Women with diabetes	
	%	n	%	n
37	13.6	59/434	17.5	14/80
38	10.2	154/1517	22.4	37/165
39	7.9	543/6863	18.3	39/213
40	6.9	1365/19 851	15.8	38/241
41	6.6	1621/24 577	16.8	21/125
42	6.2	867/14 081	19.0	8/42
Total	6.8	4609/67 323	18.1	157/866

This knowledge is important to clinicians who provide obstetric care for women who are expecting large offspring, especially when the mother has diabetes. Our finding suggests that when a women with diabetes is carrying an offspring of high weight (≥ 4500 grams), a vaginal delivery confers a high risk of adverse outcome. In such a situation a planned cesarean should be considered, independent of length of gestation.

We have made separate analyses in women carrying an offspring weighing 4000-4500 grams. I all these pregnancies, the risk of shoulder dystocia is higher before term than at or after term, and this finding was especially pronounced among women with diabetes (Table 7).

Table 7. Absolute risk (%) of shoulder dystocia among women without and with diabetes expecting an offspring weighing 4000-4499 grams according to pregnancy week at delivery

	Shoulder dystocia			
	Women without diabetes		Women with diabetes	
	%	n	%	n
Pregnancy week at delivery				
37	3.0	76/2511	10.4	21/202
38	2.6	280/10 913	9.4	37/395
39	2.0	896/44 921	8.8	53/601
40	1.8	1803/100 269	3.5	25/705
41	1.6	1607/99 412	4.0	12/298
42	1.6	753/47 676	2.0	2/102
Total	1.8	5415/ 305 702	6.5	150/2303

There is an increasing risk of shoulder dystocia by increasing offspring birthweight, and the birthweight increases by increasing pregnancy week. Our results suggest that after adjustment for birthweight the risk of shoulder dystocia decrease by increasing pregnancy week at delivery. So, what yields the lowest risk of shoulder dystocia; induction of labor before term or wait for spontaneous labor when the offspring most likely has increased in weight? Induction of delivery yields an independent 20% increase in shoulder dystocia risk.

To illustrate possible implications of our findings for clinical practice, we have calculated risk estimates for shoulder dystocia by pregnancy week and by using our risk estimates for shoulder dystocia by pregnancy week in deliveries with and without induction of labor. We

have calculated the risk of shoulder dystocia assuming offspring weight 3800 grams at pregnancy week 37, and the weight gain per week is assumed to be the mean (in grams) as for the population as a whole for both for women with and women without diabetes (Figure 6 and Figure 7). Figure 6 shows the shoulder dystocia risk among women with diabetes. In deliveries at pregnancy week 38 after labor induction (3997 grams), there is a risk of 8.4% for shoulder dystocia. Without labour induction there may be a spontaneous delivery in pregnancy week 40 and the offspring has gained more weight (4269 grams), but still the risk of shoulder dystocia is lower (6.4%) than it would have been after labor induction in week 38.

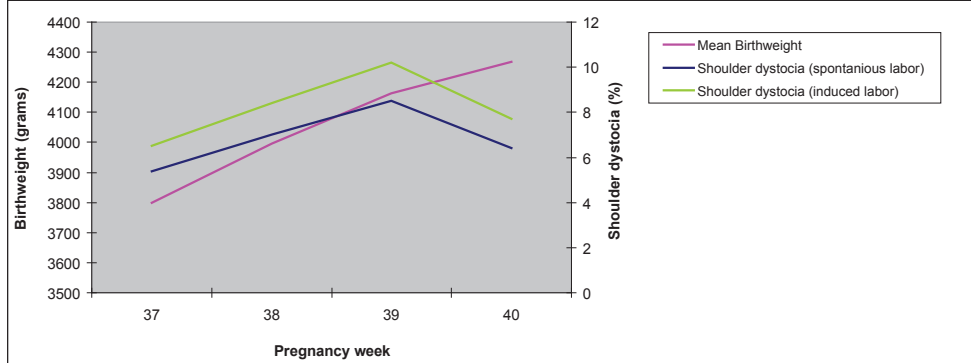


Figure 8. Risk of shoulder dystocia (%) among women with diabetes, with spontaneous labor and with induced labor, according to pregnancy week at delivery. The assumptions for offspring weight are shown in the red line

These findings suggest that induction of labor before term of women with diabetes who is carrying an offspring with moderate high weight (3800 in pregnancy week 37) is of no benefit when it comes to reducing the risk of shoulder dystocia. However, there may be other medical reasons to induce labor in women with diabetes before term.

Figure 7 shows the shoulder dystocia risk in pregnancies without diabetes. In deliveries in pregnancy week 38 after labor induction (3997 grams), there is a risk of 2.3% for shoulder dystocia. Without labour induction there may be a spontaneous delivery in pregnancy week 40 and the offspring has gained more weight (4269 grams), but still the risk of shoulder dystocia is not higher (2.2%) than it would have been after labor induction at week 38. Again, there is of no benefit to induce labor before term when it comes to reducing the risk of shoulder dystocia.

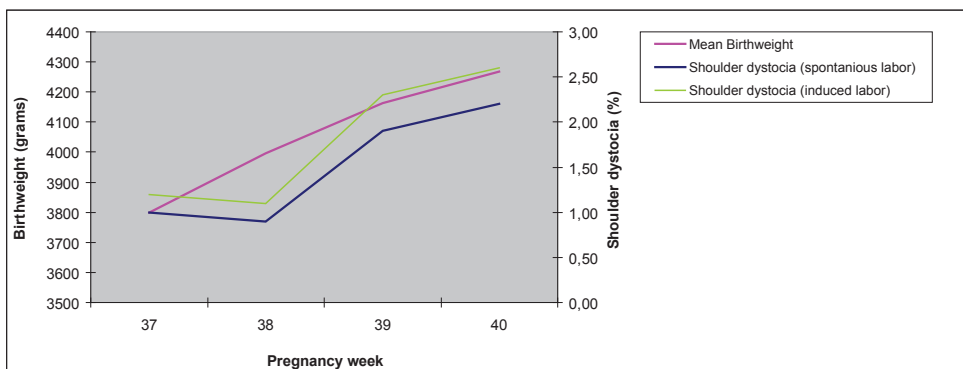


Figure 9. Risk of shoulder dystocia (%) among women without diabetes, with spontaneous labor and with induced labor, according to estimated birthweight and pregnancy week at delivery. The assumption for offspring weight is shown in the red line

To predict the risk of shoulder dystocia, it is important to be able to estimate the fetal weight with accuracy. Clinicians can estimate fetal weight by abdominal palpation, measuring the fundal height or using ultrasonographic measurements (158-161). Most studies on the reliability of birthweight prediction are based on ultrasonographic measurements employed as either one single fetal measure or combination of fetal measurements (158, 160, 161). The different ultrasonographic methods do not seem to differ substantially when it comes to prediction of fetal macrosomia (159, 161-163). Estimation of fetal weight based on clinical methods as abdominal palpation and measuring of fundal height or estimation of fetal weight by ultrasonographic methods have limited ability to predict fetal weight that are above 4000 grams. All methods suffer from false positive and false negative results (161, 163-168). There is some evidence that repeated ultrasonographic measurements may improve the predictive accuracy of fetal macrosomia (161, 163). The use of magnetic resonance imaging (MRI) of the body composition of the fetus may in the future become an alternative tool to estimate the fetal weight, but this needs further documentation (169, 170). A computerized combination of a number of risk factors together with ultrasonographic measurement of the fetal weight has also been suggested to improve the prediction of macrosomia (171).

To conclude, clinicians still have inaccurate tools for estimating fetal weight (161). In the future, methods for birthweight estimation and prediction of fetal macrosomia need to be improved and validated to provide a higher degree of accuracy for this measurement. Reliable knowledge about the fetal weight will improve the ability to give qualified advice

during clinical decision making for example to women with a history of shoulder dystocia or women with diabetes regarding mode of delivery.

Since we still are unable to make accurate estimates of fetal weight and to predict shoulder dystocia, health personnel in the maternity wards must constantly be prepared to resolve shoulder dystocia should the situation arise. Repeated emergency drills to ensure obstetric skills among midwife and obstetricians in maternity wards are absolutely necessary to resolve shoulder dystocia in the best way (88-90, 93).

7.0 CONCLUSIONS

A history of shoulder dystocia increased the risk of shoulder dystocia in the second delivery as compared to women without such a history, and 7.3% of women with a history of shoulder dystocia experienced a new shoulder dystocia. The combination of a history of shoulder dystocia and high offspring birthweight in the second pregnancy conferred a high risk of recurrence. If the birthweight was low or normal, the risk of shoulder dystocia in the second delivery was low, regardless of a history of shoulder dystocia.

High offspring birthweight was a major risk factor for shoulder dystocia and three quarters (75%) of all shoulder dystocia cases occurred in deliveries of offspring weighing more than 4000 grams. Parous women were at higher risk of shoulder dystocia compared to primiparous women, also after adjustment for offspring birthweight and other study factors. The positive association of birthweight with shoulder dystocia showed similar patterns across categories of parity, but at high birthweights parous women were at higher risk of shoulder dystocia than primiparous women.

After adjustment for offspring birthweight there was a decreasing risk of shoulder dystocia with increasing pregnancy week. This finding was particularly pronounced in diabetic pregnancies. Consequently, postterm delivery is not an independent risk factor for shoulder dystocia.

Reference List

1. Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong C, editors. Williams obstetrics. 23 ed. New York: McGraw-Hill Medical; 2010.
2. Gherman RB, Chauhan S, Ouzounian JG, Lerner H, Gonik B, Goodwin TM. Shoulder dystocia: the unpreventable obstetric emergency with empiric management guidelines. *Am J Obstet Gynecol.* 2006;195(3):657-72.
3. Pecorari D. A guest editorial from abroad: meditations on a nightmare of modern midwifery: shoulder dystocia. *Obstet Gynecol Surv.* 1999;54(6):353-4.
4. Gherman RB. Shoulder dystocia: an evidence-based evaluation of the obstetric nightmare. *Clin Obstet Gynecol.* 2002;45(2):345-62.
5. Baskett TF. Shoulder dystocia. *Best Pract Res Clin Obstet Gynaecol.* 2002;16(1):57-68.
6. Sokol RJ, Blackwell SC. ACOG practice bulletin: Shoulder dystocia. Number 40, November 2002. (Replaces practice pattern number 7, October 1997). *Int J Gynaecol Obstet.* 2003;80(1):87-92.
7. Langer O, Berkus MD, Huff RW, Samueloff A. Shoulder dystocia: should the fetus weighing greater than or equal to 4000 grams be delivered by cesarean section? *Am J Obstet Gynecol.* 1991;165(4 Pt 1):831-7.
8. Smith R. Parturition. *N Engl J Med.* 2007;356(3):271-83.
9. Benedetti TJ, Gabbe SG. Shoulder dystocia. A complication of fetal macrosomia and prolonged second stage of labor with midpelvic delivery. *Obstet Gynecol.* 1978;52(5):526-9.
10. Resnik R. Management of shoulder girdle dystocia. *Clin Obstet Gynecol.* 1980;23(2):559-64.
11. Hopwood HG, Jr. Shoulder dystocia: fifteen years' experience in a community hospital. *Am J Obstet Gynecol.* 1982;144(2):162-6.
12. Sandmire HF, Demott RK. Erb's palsy causation: a historical perspective. *Birth.* 2002;29(1):52-4.
13. Beall MH, Spong CY, Ross MG. A randomized controlled trial of prophylactic maneuvers to reduce head-to-body delivery time in patients at risk for shoulder dystocia. *Obstet Gynecol.* 2003;102(1):31-5.

14. Gherman RB, Goodwin TM, Souter I, Neumann K, Ouzounian JG, Paul RH. The McRoberts' maneuver for the alleviation of shoulder dystocia: how successful is it? *Am J Obstet Gynecol.* 1997;176(3):656-61.
15. Gherman RB, Ouzounian JG, Goodwin TM. Obstetric maneuvers for shoulder dystocia and associated fetal morbidity. *Am J Obstet Gynecol.* 1998;178(6):1126-30.
16. McFarland M, Hod M, Piper JM, Xenakis EM, Langer O. Are labor abnormalities more common in shoulder dystocia? *Am J Obstet Gynecol.* 1995;173(4):1211-4.
17. Spong CY, Beall M, Rodrigues D, Ross MG. An objective definition of shoulder dystocia: prolonged head-to-body delivery intervals and/or the use of ancillary obstetric maneuvers. *Obstet Gynecol.* 1995;86(3):433-6.
18. Beall MH, Spong C, McKay J, Ross MG. Objective definition of shoulder dystocia: a prospective evaluation. *Am J Obstet Gynecol.* 1998;179(4):934-7.
19. Gurewitsch ED, Allen RH. Reducing the risk of shoulder dystocia and associated brachial plexus injury. *Obstet Gynecol Clin North Am.* 2011;38(2):247-69, x.
20. Mollberg M, Wennergren M, Bager B, Ladfors L, Hagberg H. Obstetric brachial plexus palsy: a prospective study on risk factors related to manual assistance during the second stage of labor. *Acta Obstet Gynecol Scand.* 2007;86(2):198-204.
21. Locatelli A, Incerti M, Ghidini A, Longoni A, Casarico G, Ferrini S, et al. Head-to-body delivery interval using 'two-step' approach in vaginal deliveries: effect on umbilical artery pH. *J Matern Fetal Neonatal Med.* 2011;24(6):799-803.
22. Bourgeois ML. Observations diverses sur la stérilité, perte fruit, faecundité, accouchemens et maladies des femmes et des nouveau naiz. Paris: Jean Dehoury; 1652.
23. Beer E. History of extraction of the posterior arm to resolve shoulder dystocia. *Obstet Gynecol Surv.* 2006;61(3):149-51.
24. Smellie W. Smellie's Treatise on the theory and practice of midwifery. McClintock A, editor. London: The New Sydenham Society; 1876.
25. Barnum CG, Groton C. Dystocia due to the shoulders. *Am J Obstet Gynecol.* 1945;50:439-42.
26. Gonik B, Stringer CA, Held B. An alternative maneuver for management of shoulder dystocia. *Am J Obstet Gynecol.* 1983;145:882-4.
27. Woods CE, Westbury NY. A principle of physics as applicable to shoulder delivery. *Am J Obstet Gynecol.* 1942;45:796-804.

28. Løvset J. Vaginal operative delivery. Oslo: Universitetsforlaget; 1968.
29. Løvset J, Brandstrup E. Lærebok i obstetikk for jordmødre. Oslo: Aschehoug; 1959.
30. Swarts DP. Shoulder girdle dystocia in vertex delivery: clinical study and review. *Obstet Gynecol.* 1960;15:194-206.
31. Acker DB, Sachs BP, Friedman EA. Risk factors for shoulder dystocia. *Obstet Gynecol.* 1985;66(6):762-8.
32. Gross SJ, Shime J, Farine D. Shoulder dystocia: predictors and outcome. A five-year review. *Am J Obstet Gynecol.* 1987;156(2):334-6.
33. Nocon JJ, McKenzie DK, Thomas LJ, Hansell RS. Shoulder dystocia: an analysis of risks and obstetric maneuvers. *Am J Obstet Gynecol.* 1993;168(6 Pt 1):1732-7.
34. Allen RH, Bankoski BR, Nagey DA. Simulating birth to investigate clinician-applied loads on newborns. *Med Eng Phys.* 1995;17(5):380-4.
35. Geary M, McParland P, Johnson H, Stronge J. Shoulder dystocia--is it predictable? *Eur J Obstet Gynecol Reprod Biol.* 1995;62(1):15-8.
36. Nesbitt TS, Gilbert WM, Herrchen B. Shoulder dystocia and associated risk factors with macrosomic infants born in California. *Am J Obstet Gynecol.* 1998;179(2):476-80.
37. Kees S, Margalit V, Schiff E, Mashiach S, Carp HJ. Features of shoulder dystocia in a busy obstetric unit. *J Reprod Med.* 2001;46(6):583-8.
38. Stallings SP, Edwards RK, Johnson JW. Correlation of head-to-body delivery intervals in shoulder dystocia and umbilical artery acidosis. *Am J Obstet Gynecol.* 2001;185(2):268-74.
39. Al HM, Geary M, Byrne P, McKenna P. Shoulder dystocia: risk factors and maternal and perinatal outcome. *J Obstet Gynaecol.* 2001;21(4):352-4.
40. Christofferson M, Rydhstroem H. Shoulder dystocia and brachial plexus injury: a population-based study. *Gynecol Obstet Invest.* 2002;53(1):42-7.
41. Ouzounian JG, Gherman RB. Shoulder dystocia: are historic risk factors reliable predictors? *Am J Obstet Gynecol.* 2005;192(6):1933-5.
42. MacKenzie IZ, Shah M, Lean K, Dutton S, Newdick H, Tucker DE. Management of shoulder dystocia: trends in incidence and maternal and neonatal morbidity. *Obstet Gynecol.* 2007;110(5):1059-68.
43. Christie LR, Harriott JA, Mitchell SY, Fletcher HM, Bambury IG. Shoulder dystocia in a Jamaican cohort. *Int J Gynaecol Obstet.* 2009;104(1):25-7.

44. Moore HM, Reed SD, Batra M, Schiff MA. Risk factors for recurrent shoulder dystocia, Washington state, 1987-2004. *Am J Obstet Gynecol.* 2008;198(5):e16-e24.
45. Fadl HE, Ostlund IK, Magnuson AF, Hanson US. Maternal and neonatal outcomes and time trends of gestational diabetes mellitus in Sweden from 1991 to 2003. *Diabet Med.* 2010;27(4):436-41.
46. Dodd JM, Catcheside B, Scheil W. Can shoulder dystocia be reliably predicted? *Aust N Z J Obstet Gynaecol.* 2012;52(3):248-52.
47. Richter HE, Brumfield CG, Cliver SP, Burgio KL, Neely CL, Varner RE. Risk factors associated with anal sphincter tear: a comparison of primiparous patients, vaginal births after cesarean deliveries, and patients with previous vaginal delivery. *Am J Obstet Gynecol.* 2002;187(5):1194-8.
48. el Madany AA, Jallad KB, Radi FA, el HH, O'deh HM. Shoulder dystocia: anticipation and outcome. *Int J Gynaecol Obstet.* 1991;34(1):7-12.
49. Mazouni C, Menard JP, Porcu G, Cohen-Solal E, Heckenroth H, Gamberre M, et al. Maternal morbidity associated with obstetrical maneuvers in shoulder dystocia. *Eur J Obstet Gynecol Reprod Biol.* 2006;129(1):15-8.
50. Gherman RB, Ouzounian JG, Incerpi MH, Goodwin TM. Symphyseal separation and transient femoral neuropathy associated with the McRoberts' maneuver. *Am J Obstet Gynecol.* 1998;178(3):609-10.
51. Leung TY, Chung TK. Severe chronic morbidity following childbirth. *Best Pract Res Clin Obstet Gynaecol.* 2009;23(3):401-23.
52. Hope P, Breslin S, Lamont L, Lucas A, Martin D, Moore I, et al. Fatal shoulder dystocia: a review of 56 cases reported to the Confidential Enquiry into Stillbirths and Deaths in Infancy. *BJOG.* 1998;105(12):1256-61.
53. Mollberg M, Hagberg H, Bager B, Lilja H, Ladfors L. High birthweight and shoulder dystocia: the strongest risk factors for obstetrical brachial plexus palsy in a Swedish population-based study. *Acta Obstet Gynecol Scand.* 2005;84(7):654-9.
54. Iffy L, Brimacombe M, Apuzzio JJ, Varadi V, Portuondo N, Nagy B. The risk of shoulder dystocia related permanent fetal injury in relation to birth weight. *Eur J Obstet Gynecol Reprod Biol.* 2008;136(1):53-60.
55. Raio L, Ghezzi F, Di NE, Buttarelli M, Franchi M, Durig P, et al. Perinatal outcome of fetuses with a birth weight greater than 4500 g: an analysis of 3356 cases. *Eur J Obstet Gynecol Reprod Biol.* 2003;109(2):160-5.

56. Sacks DA, Chen W. Estimating fetal weight in the management of macrosomia. *Obstet Gynecol Surv.* 2000;55(4):229-39.
57. Christoffersson M, Kannisto P, Rydhstroem H, Stale H, Walles B. Shoulder dystocia and brachial plexus injury: a case-control study. *Acta Obstet Gynecol Scand.* 2003;82(2):147-51.
58. Mollberg M, Lagerkvist AL, Johansson U, Bager B, Johansson A, Hagberg H. Comparison in obstetric management on infants with transient and persistent obstetric brachial plexus palsy. *J Child Neurol.* 2008;23(12):1424-32.
59. Gonik B, Zhang N, Grimm MJ. Defining forces that are associated with shoulder dystocia: the use of a mathematic dynamic computer model. *Am J Obstet Gynecol.* 2003;188(4):1068-72.
60. Gurewitsch ED, Kim EJ, Yang JH, Outland KE, McDonald MK, Allen RH. Comparing McRoberts' and Rubin's maneuvers for initial management of shoulder dystocia: an objective evaluation. *Am J Obstet Gynecol.* 2005;192(1):153-60.
61. Keller JD, Lopez-Zeno JA, Dooley SL, Socol ML. Shoulder dystocia and birth trauma in gestational diabetes: a five-year experience. *Am J Obstet Gynecol.* 1991;165(4 Pt 1):928-30.
62. Ecker JL, Greenberg JA, Norwitz ER, Nadel AS, Repke JT. Birth weight as a predictor of brachial plexus injury. *Obstet Gynecol.* 1997;89(5 Pt 1):643-7.
63. Foad SL, Mehlman CT, Ying J. The epidemiology of neonatal brachial plexus palsy in the United States. *J Bone Joint Surg Am.* 2008;90(6):1258-64.
64. Evans-Jones G, Kay SP, Weindling AM, Cranny G, Ward A, Bradshaw A, et al. Congenital brachial palsy: incidence, causes, and outcome in the United Kingdom and Republic of Ireland. *Arch Dis Child Fetal Neonatal Ed.* 2003;88(3):F185-F9.
65. Leung T, Stuart O, Sahota D, Suen S, Lau T, Lao T. Head-to-body delivery interval and risk of fetal acidosis and hypoxic ischaemic encephalopathy in shoulder dystocia: a retrospective review. *BJOG.* 2010.
66. Heazell AE, Judge JK, Bhatti NR. A retrospective study to determine if umbilical cord pH correlates with duration of delay between delivery of the head and body in shoulder dystocia. *J Obstet Gynaecol.* 2004;24(7):776-7.
67. RCOG. Royal College of Obstetricians and Gynaecologists. Shoulder Dystocia. Green-top 42. *BJOG.* 2012.
68. Wood C, Ng KH, Hounslow D, Benning H. Time--an important variable in normal delivery. *J Obstet Gynaecol Br Commonw.* 1973;80(4):295-300.

69. Lerner H, Durlacher K, Smith S, Hamilton E. Relationship between head-to-body delivery interval in shoulder dystocia and neonatal depression. *Obstet Gynecol.* 2011;118(2 Pt 1):318-22.
70. Gonen R, Spiegel D, Abend M. Is macrosomia predictable, and are shoulder dystocia and birth trauma preventable? *Obstet Gynecol.* 1996;88(4 Pt 1):526-9.
71. Thangaratinam S, Rogozinska E, Jolly K, Glinkowski S, Roseboom T, Tomlinson JW, et al. Effects of interventions in pregnancy on maternal weight and obstetric outcomes: meta-analysis of randomised evidence. *BMJ.* 2012;344:e2088.
72. Crowther CA, Hiller JE, Moss JR, McPhee AJ, Jeffries WS, Robinson JS, et al. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. *N Engl J Med.* 2005;352(24):2477-86.
73. Landon MB, Spong CY, Thom E, Carpenter MW, Ramin SM, Casey B, et al. A multicenter, randomized trial of treatment for mild gestational diabetes. *N Engl J Med.* 2009;361(14):1339-48.
74. Rae A, Bond D, Evans S, North F, Roberman B, Walters B. A randomised controlled trial of dietary energy restriction in the management of obese women with gestational diabetes. *Aust N Z J Obstet Gynaecol.* 2000;40(4):416-22.
75. Jeffries K, Shub A, Walker SP, Hiscock R, Permezel M. Reducing excessive weight gain in pregnancy: a randomised controlled trial. *Med J Aust.* 2009;191(8):429-33.
76. Horvath K, Koch K, Jeitler K, Matyas E, Bender R, Bastian H, et al. Effects of treatment in women with gestational diabetes mellitus: systematic review and meta-analysis. *BMJ.* 2010;340:c1395.
77. Boulvain M, Stan C, Irion O. Elective delivery in diabetic pregnant women. *Cochrane database of systematic reviews.* 2001(2):CD001997.
78. Kjos SL, Henry OA, Montoro M, Buchanan TA, Mestman JH. Insulin-requiring diabetes in pregnancy: a randomized trial of active induction of labor and expectant management. *Am J Obstet Gynecol.* 1993;169(3):611-5.
79. Chauhan SP, Grobman WA, Gherman RA, Chauhan VB, Chang G, Magann EF, et al. Suspicion and treatment of the macrosomic fetus: a review. *Am J Obstet Gynecol.* 2005;193(2):332-46.
80. Sanchez-Ramos L, Bernstein S, Kaunitz AM. Expectant management versus labor induction for suspected fetal macrosomia: a systematic review. *Obstet Gynecol.* 2002;100(5 Pt 1):997-1002.

81. Irion O, Boulvain M. Induction of labour for suspected fetal macrosomia. Cochrane database of systematic reviews. 2000(2):CD000938.
82. Rouse DJ, Owen J. Prophylactic cesarean delivery for fetal macrosomia diagnosed by means of ultrasonography--A Faustian bargain? *Am J Obstet Gynecol.* 1999;181(2):332-8.
83. Conway DL, Langer O. Elective delivery of infants with macrosomia in diabetic women: reduced shoulder dystocia versus increased cesarean deliveries. *Am J Obstet Gynecol.* 1998;178(5):922-5.
84. Poggi SH, Allen RH, Patel CR, Ghidini A, Pezzullo JC, Spong CY. Randomized trial of McRoberts versus lithotomy positioning to decrease the force that is applied to the fetus during delivery. *Am J Obstet Gynecol.* 2004;191(3):874-8.
85. Athukorala C, Middleton P, Crowther CA. Intrapartum interventions for preventing shoulder dystocia. Cochrane database of systematic reviews. 2006(4):CD005543.
86. Allen RH. On the mechanical aspects of shoulder dystocia and birth injury. *Clin Obstet Gynecol.* 2007;50(3):607-23.
87. Allen RH, Bankoski BR, Butzin CA, Nagey DA. Comparing clinician-applied loads for routine, difficult, and shoulder dystocia deliveries. *BJOG.* 1994;171(6):1621-7.
88. Crofts JF, Ellis D, Draycott TJ, Winter C, Hunt LP, Akande VA. Change in knowledge of midwives and obstetricians following obstetric emergency training: a randomised controlled trial of local hospital, simulation centre and teamwork training. *BJOG.* 2007;114(12):1534-41.
89. Crofts JF, Bartlett C, Ellis D, Hunt LP, Fox R, Draycott TJ. Management of shoulder dystocia: skill retention 6 and 12 months after training. *Obstet Gynecol.* 2007;110(5):1069-74.
90. Sorensen JL, Lokkegaard E, Johansen M, Ringsted C, Kreiner S, McAleer S. The implementation and evaluation of a mandatory multi-professional obstetric skills training program. *Acta Obstet Gynecol Scand.* 2009;88(10):1107-17.
91. Goffman D, Heo H, Pardanani S, Merkatz IR, Bernstein PS. Improving shoulder dystocia management among resident and attending physicians using simulations. *Am J Obstet Gynecol.* 2008;199(3):294-5.
92. Deering S, Poggi S, Macedonia C, Gherman R, Satin AJ. Improving resident competency in the management of shoulder dystocia with simulation training. *Obstet Gynecol.* 2004;103(6):1224-8.

93. Draycott TJ, Crofts JF, Ash JP, Wilson LV, Yard E, Sibanda T, et al. Improving neonatal outcome through practical shoulder dystocia training. *Obstet Gynecol.* 2008;112(1):14-20.
94. Grobman WA, Miller D, Burke C, Hornbogen A, Tam K, Costello R. Outcomes associated with introduction of a shoulder dystocia protocol. *Am J Obstet Gynecol.* 2011;205(6):513-7.
95. Rubin A. Management of shoudler dystocia. *JAMA.* 1964;189:835-7.
96. Leung TY, Stuart O, Suen SS, Sahota DS, Lau TK, Lao TT. Comparison of perinatal outcomes of shoulder dystocia alleviated by different type and sequence of manoeuvres: a retrospective review. *BJOG.* 2011;118(8):985-90.
97. Hoffman MK, Bailit JL, Branch DW, Burkman RT, Van VP, Lu L, et al. A Comparison of Obstetric Maneuvers for the Acute Management of Shoulder Dystocia. *Obstet Gynecol.* 2011;117(6):1272-8.
98. Poggi SH, Spong CY, Allen RH. Prioritizing posterior arm delivery during severe shoulder dystocia. *Obstet Gynecol.* 2003;101(5 Pt 2):1068-72.
99. Baskett TF, Allen AC. Perinatal implications of shoulder dystocia. *Obstet Gynecol.* 1995;86(1):14-7.
100. Paris AE, Greenberg JA, Ecker JL, McElrath TF. Is an episiotomy necessary with a shoulder dystocia? *Am J Obstet Gynecol.* 2011;205(3):217-3.
101. Gurewitsch ED, Donithan M, Stallings SP, Moore PL, Agarwal S, Allen LM, et al. Episiotomy versus fetal manipulation in managing severe shoulder dystocia: a comparison of outcomes. *Am J Obstet Gynecol.* 2004;191(3):911-6.
102. Bruner JP, Drummond SB, Meenan AL, Gaskin IM. All-fours maneuver for reducing shoulder dystocia during labor. *J Reprod Med.* 1998;43(5):439-43.
103. Hartfield VJ. Symphysiotomy for shoulder dystocia. *Am J Obstet Gynecol.* 1986;155(1):228.
104. Wykes CB, Johnston TA, Paterson-Brown S, Johanson RB. Symphysiotomy: a lifesaving procedure. *BJOG.* 2003;110(2):219-21.
105. Goodwin TM, Banks E, Millar LK, Phelan JP. Catastrophic shoulder dystocia and emergency symphysiotomy. *Am J Obstet Gynecol.* 1997;177(2):463-4.
106. Sandberg EC. The Zavanelli maneuver: 12 years of recorded experience. *Obstet Gynecol.* 1999;93(2):312-7.

107. O'Leary JA. Cephalic replacement for shoulder dystocia: present status and future role of the Zavanelli maneuver. *Obstet Gynecol.* 1993;82(5):847-50.
108. Spellacy WN. The Zavanelli maneuver for fetal shoulder dystocia. Three cases with poor outcomes. *J Reprod Med.* 1995;40(7):543-4.
109. Rothman KJ. *Epidemiology: an introduction.* Oxford: Oxford University Press; 2002.
110. Athukorala C, Crowther CA, Willson K. Women with gestational diabetes mellitus in the ACHOIS trial: risk factors for shoulder dystocia. *Aust N Z J Obstet Gynaecol.* 2007;47(1):37-41.
111. Casey BM, Lucas MJ, McIntire DD, Leveno KJ. Pregnancy outcomes in women with gestational diabetes compared with the general obstetric population. *Obstet Gynecol.* 1997;90(6):869-73.
112. Modanlou HD, Komatsu G, Dorchester W, Freeman RK, Bosu SK. Large-for-gestational-age neonates: anthropometric reasons for shoulder dystocia. *Obstet Gynecol.* 1982;60(4):417-23.
113. McFarland MB, Trylovich CG, Langer O. Anthropometric differences in macrosomic infants of diabetic and nondiabetic mothers. *J Matern Fetal Med.* 1998;7(6):292-5.
114. Kitzmiller JL, Mall JC, Gin GD, Hendricks SK, Newman RB, Scheerer L. Measurement of fetal shoulder width with computed tomography in diabetic women. *Obstet Gynecol.* 1987;70(6):941-5.
115. Mintz MC, Landon MB, Gabbe SG, Marinelli DL, Ludmir J, Grumbach K, et al. Shoulder soft tissue width as a predictor of macrosomia in diabetic pregnancies. *Am J Perinatol.* 1989;6(2):240-3.
116. Stotland NE, Caughey AB, Breed EM, Escobar GJ. Risk factors and obstetric complications associated with macrosomia.[Erratum appears in *Int J Gynaecol Obstet.* 2005 Jul;90(1):88]. *Int J Gynaecol Obstet.* 2004;87(3):220-6.
117. Mazouni C, Porcu G, Cohen-Solal E, Heckenroth H, Guidicelli B, Bonnier P, et al. Maternal and anthropomorphic risk factors for shoulder dystocia. *Acta Obstet Gynecol Scand.* 2006;85(5):567-70.
118. Gemer O, Bergman M, Segal S. Labor abnormalities as a risk factor for shoulder dystocia. *Acta Obstet Gynecol Scand.* 1999;78(8):735-6.
119. Sama JC, Iffy L. Maternal weight and fetal injury at birth: data deriving from medico-legal research. *Med Law.* 1998;17(1):61-8.

120. Robinson H, Tkatch S, Mayes DC, Bott N, Okun N. Is maternal obesity a predictor of shoulder dystocia? *Obstet Gynecol.* 2003;101(1):24-7.
121. Leung TY, Leung TN, Sahota DS, Chan OK, Chan LW, Fung TY, et al. Trends in maternal obesity and associated risks of adverse pregnancy outcomes in a population of Chinese women. *BJOG.* 2008;115(12):1529-37.
122. Jensen DM, Damm P, Sorensen B, Molsted-Pedersen L, Westergaard JG, Ovesen P, et al. Pregnancy outcome and prepregnancy body mass index in 2459 glucose-tolerant Danish women. *Am J Obstet Gynecol.* 2003;189(1):239-44.
123. Cedergren MI. Maternal morbid obesity and the risk of adverse pregnancy outcome. *Obstet Gynecol.* 2004;103(2):219-24.
124. Gilead R, Yaniv Salem S, Sergienko R, Sheiner E. Maternal "isolated" obesity and obstetric complications. *J Matern Fetal Neonatal Med.* 2012;25(12):2579-82.
125. Wilcox MA, Chang AM, Johnson IR. The effects of parity on birthweight using successive pregnancies. *Acta Obstet Gynecol Scand.* 1996;75(5):459-3.
126. Gunderson EP, Abrams B. Epidemiology of gestational weight gain and body weight changes after pregnancy. *Epidemiol Rev.* 2000;22(2):261-74.
127. Lee SK, Sobal J, Frongillo EA, Olson CM, Wolfe WS. Parity and body weight in the United States: differences by race and size of place of residence. *Obes Res.* 2005;13(7):1263-9.
128. Ohlin A, Rossner S. Maternal body weight development after pregnancy. *Int J Gynaecol Obstet.* 1990;14(2):159-73.
129. Rossner S, Ohlin A. Pregnancy as a risk factor for obesity: lessons from the Stockholm Pregnancy and Weight Development Study. *Obes Res.* 1995;3 Suppl 2:267s-75s.
130. Sandmire HF, O'Hallain TJ. Shoulder dystocia: its incidence and associated risk factors. *Int J Gynaecol Obstet.* 1988;26(1):65-73.
131. Gross TL, Sokol RJ, Williams T, Thompson K. Shoulder dystocia: a fetal-physician risk. *Am J Obstet Gynecol.* 1987;156(6):1408-18.
132. Rydhstrom H, Ingemarsson I. The extremely large fetus--antenatal identification, risks, and proposed management. *Acta Obstet Gynecol Scand.* 1989;68(1):59-63.
133. Lewis DF, Edwards MS, Asrat T, Adair CD, Brooks G, London S. Can shoulder dystocia be predicted? Preconceptive and prenatal factors. *J Reprod Med.* 1998;43(8):654-8.

134. Sheiner E, Levy A, HersHKovitz R, Hallak M, Hammel RD, Katz M, et al. Determining factors associated with shoulder dystocia: a population-based study. *Eur J Obstet Gynecol Reprod Biol.* 2006;126(1):11-5.
135. Mansor A, Arumugam K, Omar SZ. Macrosomia is the only reliable predictor of shoulder dystocia in babies weighing 3.5 kg or more. *Eur J Obstet Gynecol Reprod Biol.* 2010;149(1):44-6.
136. Gupta M, Hockley C, Quigley MA, Yeh P, Impey L. Antenatal and intrapartum prediction of shoulder dystocia. *Eur J Obstet Gynecol Reprod Biol.* 2010;151(2):134-9.
137. Bjorstad AR, Irgens-Hansen K, Daltveit AK, Irgens LM. Macrosomia: mode of delivery and pregnancy outcome. *Acta Obstet Gynecol Scand.* 2010;89(5):664-9.
138. Vidarsdottir H, Geirsson RT, Hardardottir H, Valdimarsdottir U, Dagbjartsson A. Obstetric and neonatal risks among extremely macrosomic babies and their mothers. *Am J Obstet Gynecol.* 2011;204(5):423-6.
139. Tsur A, Sergienko R, Wiznitzer A, Zlotnik A, Sheiner E. Critical analysis of risk factors for shoulder dystocia. *Arch Gynecol Obstet.* 2012;285(5):1225-9.
140. Acker DB, Sachs BP, Friedman EA. Risk factors for shoulder dystocia in the average-weight infant. *Obstet Gynecol.* 1986;67(5):614-8.
141. Poggi SH, Stallings SP, Ghidini A, Spong CY, Deering SH, Allen RH. Intrapartum risk factors for permanent brachial plexus injury. *Am J Obstet Gynecol.* 2003;189(3):725-9.
142. Mehta SH, Bujold E, Blackwell SC, Sorokin Y, Sokol RJ. Is abnormal labor associated with shoulder dystocia in nulliparous women? *Am J Obstet Gynecol.* 2004;190(6):1604-7.
143. Lurie S, Levy R, Ben-Arie A, Hagay Z. Shoulder dystocia: could it be deduced from the labor partogram? *Am J Perinatol.* 1995;12(1):61-2.
144. Demissie K, Rhoads GG, Smulian JC, Balasubramanian BA, Gandhi K, Joseph KS, et al. Operative vaginal delivery and neonatal and infant adverse outcomes: population based retrospective analysis. *BMJ.* 2004;329(7456):24-9.
145. Caughey AB, Sandberg PL, Zlatnik MG, Thiet MP, Parer JT, Laros RK, Jr. Forceps compared with vacuum: rates of neonatal and maternal morbidity. *Obstet Gynecol.* 2005;106(5 Pt 1):908-12.
146. Bofill JA, Rust OA, Devidas M, Roberts WE, Morrison JC, Martin JN, Jr. Shoulder dystocia and operative vaginal delivery. *J Matern Fetal Med.* 1997;6(4):220-4.

147. O'Leary JA. Shoulder Dystocia and Birth Injury: Prevention and Treatment. Totowa, NJ: Humana Press; 2009.
148. Lewis DF, Raymond RC, Perkins MB, Brooks GG, Heymann AR. Recurrence rate of shoulder dystocia. *Am J Obstet Gynecol.* 1995;172(5):1369-71.
149. Mehta SH, Blackwell SC, Chadha R, Sokol RJ. Shoulder dystocia and the next delivery: outcomes and management. *J Matern Fetal Neonatal Med.* 2007;20(10):729-33.
150. Ginsberg NA, Moisisdis C. How to predict recurrent shoulder dystocia. *Am J Obstet Gynecol.* 2001;184(7):1427-9.
151. Mocanu EV, Greene RA, Byrne BM, Turner MJ. Obstetric and neonatal outcome of babies weighing more than 4.5 kg: an analysis by parity. *Eur J Obstet Gynecol Reprod Biol.* 2000;92(2):229-33.
152. Revicky V, Mukhopadhyay S, Morris EP, Nieto JJ. Can we predict shoulder dystocia? *Arch Gynecol Obstet.* 2011.
153. Overland EA, Spydsaug A, Nielsen CS, Eskild A. Risk of shoulder dystocia in second delivery: does a history of shoulder dystocia matter? *Am J Obstet Gynecol.* 2009;200(5):506.e1-6.
154. Campbell MK, Ostbye T, Irgens LM. Post-term birth: risk factors and outcomes in a 10-year cohort of Norwegian births. *Obstet Gynecol.* 1997;89(4):543-8.
155. Irgens LM. The Medical Birth Registry of Norway. Epidemiological research and surveillance throughout 30 years. *Acta Obstet Gynecol Scand.* 2000;79(6):435-9.
156. Juul S. *Epidemiologi og evidens.* København: Munksgaard; 2012.
157. Rothman KJ, Greenland S, Lash TL, Buehler JW, Cahill J, Glymour MM, et al. *Modern epidemiology.* Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2008.
158. Hadlock FP, Harrist RB, Sharman RS, Deter RL, Park SK. Estimation of fetal weight with the use of head, body, and femur measurements--a prospective study. *Am J Obstet Gynecol.* 1985;151(3):333-7.
159. Chauhan SP, Hendrix NW, Magann EF, Morrison JC, Kenney SP, Devoe LD. Limitations of clinical and sonographic estimates of birth weight: experience with 1034 parturients. *Obstet Gynecol.* 1998;91(1):72-7.
160. Combs CA, Jaekle RK, Rosenn B, Pope M, Miodovnik M, Siddiqi TA. Sonographic estimation of fetal weight based on a model of fetal volume. *Obstet Gynecol.* 1993;82(3):365-70.

161. Henriksen T. The macrosomic fetus: a challenge in current obstetrics. *Acta Obstet Gynecol Scand.* 2008;87(2):134-45.
162. Coomarasamy A, Connock M, Thornton J, Khan KS. Accuracy of ultrasound biometry in the prediction of macrosomia: a systematic quantitative review. *BJOG.* 2005;112(11):1461-6.
163. O'Reilly-Green C, Divon M. Sonographic and clinical methods in the diagnosis of macrosomia. *Clin Obstet Gynecol.* 2000;43(2):309-20.
164. Melamed N, Yogev Y, Meizner I, Mashiach R, Ben-Haroush A. Sonographic prediction of fetal macrosomia: the consequences of false diagnosis. *J Ultrasound Med.* 2010;29(2):225-30.
165. Lurie S, Zalel Y, Hagay ZJ. The evaluation of accelerated fetal growth. *Curr Opin Obstet Gynecol.* 1995;7(6):477-81.
166. Skrastad RB, Eik-Nes SH, Sviggum O, Johansen OJ, Salvesen KA, Romundstad PR, et al. A randomized controlled trial of third-trimester routine ultrasound in a non-selected population. *Acta Obstet Gynecol Scand.* 2013;92(12):1353-60.
167. Bricker L, Neilson JP, Dowswell T. Routine ultrasound in late pregnancy (after 24 weeks' gestation). *Cochrane database of systematic reviews.* 2008(4):CD001451.
168. Sandmire HF. Whither ultrasonic prediction of fetal macrosomia? *Obstet Gynecol.* 1993;82(5):860-2.
169. Zaretsky MV, Reichel TF, McIntire DD, Twickler DM. Comparison of magnetic resonance imaging to ultrasound in the estimation of birth weight at term. *Am J Obstet Gynecol.* 2003;189(4):1017-20.
170. Duncan KR, Issa B, Moore R, Baker PN, Johnson IR, Gowland PA. A comparison of fetal organ measurements by echo-planar magnetic resonance imaging and ultrasound. *BJOG.* 2005;112(1):43-9.
171. Sokol RJ, Chik L, Dombrowski MP, Zador IE. Correctly identifying the macrosomic fetus: improving ultrasonography-based prediction. *Am J Obstet Gynecol.* 2000;182(6):1489-95.

